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Fish-Borne and Type E Botulism: Two Cases Due to Home-Pickled Herring

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ALTHOUGH botulism appears to be a very rare disease in this country, the likelihood that unrecognized instances occur has been strengthened by the identification of a third episode in British Columbia within a period of roughly five years. This new occurrence brings the total recorded outbreaks in Canada to 6, with 38 persons in all affected, of whom 22 died. As we have pointed out before, this is a disproportionately low incidence by comparison with the figures for the United States, as compiled by Meyer and Eddie (1), which indicate a total of 477 outbreaks, with 1281 cases, including 829 fatalities, during the period 1899-1949.

The three British Columbia episodes have involved 7 cases, 6 of them fatal, and are the only Canadian outbreaks of botulism in which the causal micro-organisms have been isolated. In 1944, at Nanaimo, B.C., two adults and a child died after consuming improperly home-canned salmon. *Cl. botulinum*, type E, was isolated from companion cans of salmon and chicken, and also from the soil of a chicken run in the garden (2). In 1948, at Grand Forks, B.C., two adults died after eating asparagus canned very carelessly on a farm. *Cl. botulinum*, type A, was in this instance isolated from two similar cans of asparagus, and from the soil of the asparagus bed (3). The present report is concerned with one fatal and one non-fatal case of botulism, which occurred

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in 1949 at Vancouver, B.C., after consumption of home-pickled herring. A type E strain was again implicated, being isolated from a small remnant of the herring, and from stomach and jejunal contents taken post-mortem.

Previous Outbreaks of Type E Botulism

Before the significant features of this recent incident are described, a few introductory comments seem relevant on fish-borne and also on type E botulism. In Table I are set forth the known type E outbreaks.

TABLE I

KNOWN OUTBREAKS OF TYPE E BOTULISM

YEAR	REPORTED BY	PLACE OF OCCURRENCE	INCRIMINATED FOOD	CASES	DEATHS
Before 1936	Gunnison, Cummings and Meyer (1936)	Soviet Ukraine	Russian sturgeon muscles and intestines	Unknown: probably many	
1934	Hazen (1937)	Westchester County, New York	German canned sprats	3	1
1934	Hazen (1938)	Cooperstown, New York	Labrador smoked salmon	3	1
1941	Geiger (1941)	San Francisco, California	Yugoslav mushrooms canned in California	3	1
1944	Dolman and Kerr (1947)	Nanaimo, B.C.	Home-canned salmon*	3	3
1949	Dolman, Chang, Kerr and Shearer (1950)	Vancouver, B.C.	Home-pickled herring	2	1

*Also isolated from home-canned chicken in same household and from nearby garden soil.

The table illustrates the following points:

(i) Type E botulism had not been identified prior to 1936, when Gunnison, Cummings and Meyer (4) reported on a group of strains of *Cl. botulinum* which could not be classed with any of the previously known types—A, B, C or D. These cultures had been originally isolated from the muscles and intestines of sturgeon by Professor Bier of the Bacteriological Institute at Dniepopetrowsk, in the Russian Ukraine. (Although information is wanting on this point, the cultures were presumably isolated from fish implicated in outbreaks of botulism, which Zlatogoroff and Soloviev (4) stated, in 1927, "appeared to be spreading more and more in the Union of Socialist Soviet Republics; during the last few years there were outbreaks in groups of people in Odessa and in Kharkov.")

(ii) Excluding the unknown number of occurrences in the U.S.S.R., but including the Vancouver episode, 5 type E outbreaks have now been recorded. These have all been of relatively small magnitude, the known cases totalling only 14, of which 7 (50 per cent) were fatal. In British Columbia, of the 5 persons affected by type E botulism, 4 (80 per cent) died. These high case fatality rates are of the same general order as the 58 per cent which has prevailed in a total of 38 cases of human botulism

recorded to date in Canada, and the 65 per cent in nearly 1300 cases reported in the United States.

(iii) All 5 type E outbreaks have occurred in North America, 2 of them in New York State, 1 in California and 2 in British Columbia. Two of the outbreaks were traced to foodstuffs apparently of European origin; while in the remaining instances the incriminated food came from the extremities of Canada, viz., Labrador and British Columbia.

(iv) The preponderance of fish as the vehicle for type E botulism is noteworthy. The isolation of type E strains from canned chicken and from garden soil, and the attribution of the California outbreak to mushroom sauce, indicate that such strains can subsist, and even develop toxin, in other vehicles; but in North America, fish has been seldom implicated in botulism, and it would seem more than coincidence that some species of fish should have been involved in all save one of the known type E outbreaks.

Botulism due to Fish

In the United States, home-canned vegetables have consistently been the chief source of human botulism. Over the period 1899-1949 inclusive, according to records of 477 outbreaks compiled by Meyer and Eddie (1), string beans were involved no fewer than 98 times; corn, 49; spinach or chard, 25; beets, 24; and asparagus, 22 times. Among fruits (much less frequently implicated) the chief offenders have been olives, with 14 outbreaks; figs, 11; and tomatoes, 7. Altogether, vegetables and fruits—the great majority of them home-canned—have accounted for 358, or 75.06 per cent of the total known botulism episodes in the U.S.A. On the other hand, various kinds of fish or sea foods were identified as the cause of only 27, or 5.66 per cent of all recorded outbreaks.

In Canada, the total of known outbreaks is far too small to justify sweeping inferences; but it seems at least noteworthy that fish or fish products should have been implicated in 3 out of 6 reported episodes. Table II shows the incidence of fish-borne botulism in the United States and Canada, respectively. The contrast between these percentages would have been even more striking if one of the Canadian outbreaks, due to beets, had not involved an unusually large number of cases and deaths; and also if one of the fish-borne outbreaks which occurred in the United States, but was due to salmon caught, smoked,

TABLE II

BOTULISM DUE TO FISH AND SEA FOODS IN THE UNITED STATES AND CANADA, 1899–1949

	U.S.A.	CANADA
Total recorded outbreaks	477	6
Fish-borne outbreaks	27 (5.66%)	3 (50.0%)
Total recorded cases	1281	38
Cases due to fish	67 (5.23%)	5 (13.2%)
Total recorded deaths	829	22
Deaths due to fish	38 (4.58%)	5 (18.2%)

gauze-wrapped and paraffin-coated in Labrador (6), were transferred (as in a sense it should be) to the column of Canadian outbreaks.

Of the 27 outbreaks ascribed in the United States to fish and sea foods, 7 were traced to salmon, 7 to tuna, 5 to clams, 4 to sardines, and one each to crab, sprats, pickled mackerel and herring, and herring. Most of these products were canned, in many instances commercially. A single fatality in 1921 at San Jacinto, California, from home-canned, pickled mackerel and herring (7), was apparently due to *Cl. botulinum* type A; as were 2 fatal cases from commercially canned sardines in 1925 in Solon, Ohio (8), and 4 cases (2 fatal) from home-canned salmon in 1925 at Yakima, Washington. On the other hand, type B strains were isolated from home-canned tuna, and from Japanese commercially canned clams, which caused fatalities in California (1). As already indicated, type E strains were isolated by Hazen from German canned sprats (9) and from Labrador smoked salmon (10), which had caused outbreaks of botulism in New York State in 1934. The 3 Canadian outbreaks were all due to non-commercial products—canned salmon, salmon eggs, and pickled herring, respectively—from the first and last of which type E organisms were isolated.

In Germany, botulism has been traditionally regarded as mainly conveyed by ham, sausages and other cured or prepared meats; but smoked fish has also been recognized as potentially dangerous. Indeed, the first allusion to the causal micro-organism having been isolated from botulinogenic fish, appears in the German literature. In 1912, van Ermengem (11) referred to Madsen as having found *Cl. botulinum* in salted mackerel. According to Meyer (1) this was a type B organism. Wasmuth (12) has recently reported a series of approximately 30 cases of botulism, including 8 deaths, which had occurred in and around Kiel since the war. Most of them were apparently due to herring steeped in dilute vinegar. *Cl. botulinum* type A was isolated from the intestinal contents of 2 of the fatal cases, and in one instance from the incriminated fish.

The early Russian literature contains numerous references to outbreaks of "ichthyism", many of which are, on clinical grounds, unmistakable examples of botulism. Most of these outbreaks have been due to big fish, especially to varieties of sturgeon; but in 1883, Chugin (13) described the course of a severe illness in 3 children of a Russian-Jewish family at Rostov-on-Don, 2 of whom died on the third and fifth days respectively, after eating raw salted herrings. The symptomatology was characteristic of botulism. Incidentally, Chugin alludes to an unpublished case of poisoning due to another species of small fish (salted breams), which he had investigated in Temnikov, in 1876. Chernishev (14), in 1899, described 2 fatalities from typical botulism in young men who had eaten raw salted herring. Again, Ganelin and Billig (15) described an outbreak of fish poisoning at Odessa in the summer of 1925. Among a total of 31 cases, 20 had recovered from an acute gastro-enteritis after eating various kinds of relatively small fish, including smoked and salted herring, purchased from several different sources. The remaining 11 persons, 7 of whom died, displayed the classical symptomatology of botulism, after eating smoked herring bought from stores served by the same wholesale fish market.

Unfortunately, even when clinical and pathological data in these Russian accounts are elaborately detailed, the bacteriological information is usually

scant and liable to misinterpretation. Some of the earlier authors, such as Anrep (16), Yakovlev (17) and Arustamov (18), refer to paralysis and death of laboratory animals injected with extracts of e.g. salted sturgeon or smoked salmon, suspected of poisoning humans; but it was customary then to ascribe such phenomena to ptomaines. Even in the comparatively recent reference, by Zlatogoroff and Soloviev (5), to 20 cases of botulism, 14 of them fatal, which occurred at Kharkov in the period 1920-26, smoked or salted sturgeon were incriminated in 18 instances, and salmon in the other 2, mainly on clinical and epidemiological grounds. However, credit is due to Chugin who conceded that after the work of Selmi and others on ptomaines, "it is natural to seek these alkaloids in poisonous sausage and fish"; but added the suggestion (13 years before van Ermengem's first isolation of *Cl. botulinum*) "On the other hand, it is possible that we are here dealing with some special disease-producing lower organisms; and therefore, in future cases of poisoning, it is always desirable to use modern stains for detecting the possible presence of *schizomycetes* in poisonous sausage or fish" (13). This conjecture was verified by his countryman Konstansov (19), in 1914, who isolated from smoked white sturgeon, which had caused several typical cases of botulism in Astrakhan, an anaerobic, toxigenic, spore-bearing bacillus. He designated it *Bacillus ichthyismi*, emphasizing the possibility that it "is only a variety of *B. botulinus*, or is even identical with it."

The general lack of bacteriological data in the Russian literature on fish poisoning, led Meyer (20) to urge caution in concluding that all outbreaks reported from that country were in fact examples of botulism. Nevertheless, dietetic habits in the Soviet Union presumably still render its people especially prone to outbreaks of fish-borne botulism. An effort was made, several months ago, to obtain up-to-date information on the incidence of botulism in the U.S.S.R., and particularly on type E outbreaks. A reply is awaited. Meanwhile, it must suffice to state that the statistical evidence does suggest type E strains may have some predilection for fish. This possibility appears strengthened by certain features of the episode now to be described.

Pickled Herring Outbreak at Vancouver, B.C.

One evening, early in October, 1949, an insurance agent, aged 31, called on an acquaintance, a plasterer, aged 29, for a social and business visit. About 9 p.m. the men decided to celebrate the sale and purchase of a policy by having a tasty snack. The host's wife was out at the time, so he took down a jar of pickled herring from the kitchen cupboard. The 24-oz. jar contained about 6 small herring, each of which had broken into some 3 pieces. The jar was one half to three-quarters full of vinegar, and pieces of chopped onion and spices were floating on top. The visitor ate about 13 pieces of herring, and the host 5 pieces. They agreed the flavour was excellent, and only a few fragments of backbone were left. These were later scraped off the plates by the wife and thrown into the garbage can.

The salesman went to work next morning, having a couple of beers on the way. His appetite was good for lunch, but he vomited it up, and went home to bed in the afternoon. He again vomited after supper and complained of a

headache; so his wife sent for a doctor, who saw him late that night, i.e. about 25 hours after the meal. At this time, the patient complained chiefly of moderate headache and vague abdominal pains. His temperature and pulse were normal, no ocular abnormalities were noted, tendon and cutaneous reflexes were present, and there was nothing to suggest an acute illness. The patient had lately not been sleeping well owing to business worries, so the condition was diagnosed as an "anxiety state". The man passed a very restless night and when re-examined by the doctor at 11 a.m. next day (38 hours after consuming the herring), he was stuporose; the pupils were dilated and non-reacting to light; limb and abdominal reflexes were absent. The patient was rushed to the Vancouver General Hospital, with a provisional diagnosis of acute drug poisoning. On admission, he was unable to speak, or to move his legs, although he nodded his head in response to questions, and could move his arms. The pupils were dilated and fixed, and he appeared unable to see. The finger tips were cyanosed. He died suddenly a short while later, about 40 hours after the fatal meal.

At autopsy, the stomach, duodenum and jejunum were dilated and flabby, while the lower part of the small intestine appeared to be in spasm. About 1 pint of evil-smelling, green fluid was taken from the stomach, and a similar amount of brown, semi-faecal material from the jejunum. The large bowel and its contents appeared normal. There were no other noteworthy features.

The other victim exhibited a rather different syndrome. He was taken ill about 18 hours after eating the herring, with severe abdominal cramps, followed by diarrhoea. However, he remained at work throughout the day, and despite having no appetite, managed to eat his supper—which he promptly vomited up. That night he developed cramps in the legs, and complained of dryness of the mouth and throat, with slight dysphagia. These symptoms gradually worsened, and as he had one or two recurrences of vomiting, a doctor was called in next morning. The physical signs noted (38 hours after the meal) were general weakness, very feeble reflexes, temperature normal, pulse 100. The symptoms complained of were pains in the calves, a bloated feeling and cramps in the abdomen, loss of appetite, and dryness of nose, throat and mouth. The patient had two loose bowel movements just before being taken to the hospital, where about 3 hours previously his visitor of two nights before had died. On admission, his pulse was 100, temperature 97.4°; pupils reacted sluggishly; abdominal and cremasteric reflexes, knee and ankle jerks, and also sphincter control, were absent; and weakness of his leg muscles was apparent. Administration of 5000 units of type A botulinus antitoxin, one vial of which happened to be on hand, was advised. Efforts to procure a further supply of any type of botulinus antitoxin proved fruitless.

During the next 24 hours, the patient continued to have abdominal cramps, suffered a good deal from retching and nausea, and vomited two or three times. His legs remained weak, but showed no paralysis, and no definite cranial nerve disturbances were noted. Next morning, his condition had improved, and three days later he was well enough to go home. Convalescence was complicated by a delayed serum reaction, general weakness, and intractable diarrhoea. He did not return to work for several weeks.

Source and Method of Preparation of Herring

The fish had been caught during the summer by a local fisherman, probably in the Gulf of Georgia, and not far from Vancouver. They were obtained at the cannery docks by the plasterer's sister-in-law, who kept them at home at room temperature for twenty-four hours, and then placed them in a nearby commercial deep-freeze locker, wrapped in grease-proof paper, where they remained undisturbed for a few weeks. Six days before the fatal supper, her sister removed the package from the locker, carried it home, and left the fish to thaw out overnight on the kitchen draining board. Next morning, not more than twenty-four hours after removal of the fish from the deep-freeze, they were gutted, the heads and tails chopped off, and the skins peeled from the flesh, which was firm. The fish were then immediately packed into a 24-oz. jar, and pickling fluid poured over them until the jar was about three-quarters full. This fluid comprised equal parts of "white" vinegar and water, with added spices and chopped onion. After the top had been screwed on, the jar was placed in the kitchen cupboard, where the temperature was estimated to have averaged around 70°F.

About 4 days later, the housewife opened the jar, sampled a small piece of fish pulled from the end of one of the herring, found it tasted flat, and therefore added salt. She had previously pickled only herring which had been slightly salted prior to freezing: this particular batch had not been so treated. The jar was then restored to the shelf, where it remained untouched until removed by the husband.

Laboratory Findings

On hearing about the insurance salesman's death, the woman threw the remaining pickling fluid down the toilet, and thoroughly scoured the jar out with soapy water. Next day, in response to urgent appeals, she managed to retrieve from the garbage pail into which they had been thrown three nights before, a few small fragments of herring backbone, festooned with coffee grounds. A stained smear from a moist swab applied to an intervertebral space showed a few Gram-positive bacilli, with a tendency towards Gram-negativity and some apparent vacuolation. From a small piece of flesh teased from a vertebra, we eventually isolated a pure culture of a strain of *Cl. botulinum* type E.

Human blood agar plates were streaked from peptone beef-infusion broth, with ground meat added according to Stewart's formula (21), which had been inoculated with a fragment of fish, incubated at 37°C. for several days, and then heated in a boiling water bath for about 3 minutes only. These plates were incubated under complete anaerobiosis for 2-3 days at 37°C., and suspicious colonies then carefully picked. The task of final isolation was complicated by the relative heat-lability of type E spores; and also by the simultaneous presence of *B. subtilis*, and of numerous other spore-bearing aerobic and anaerobic bacilli, many of which may have originated from the spices used in pickling.

Similar techniques applied to neutralized samples of gastric contents removed at autopsy (original pH 3.0) yielded an apparently identical culture.

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Moreover, the presence of approximately 1000 mouse M.L.D.'s of type E toxin was demonstrated per cc. of jejunal contents (pH 5.5), and from this fluid a type E strain was likewise isolated. Repeated efforts to detect *Cl. botulinum* in the stomach washings of the recovered case proved unavailing.

Main Characteristics of Type E ("VH") Strain Isolated

In stained preparations, the Vancouver herring ("VH") strain was a Gram-positive, round-ended bacillus of 4-6μ average length, with tendencies towards vacuolation and Gram-negativity. Spore formation was erratic, and appeared to be associated with exuberant rather than restricted growth; but when present, the spores were large, oval and subterminal. In fresh hanging-drop preparations, the bacilli slowly wobbled across the field; but most of them soon ceased to be mobile, became indistinct in outline, and appeared to be undergoing some form of autolysis. The peptone beef-infusion broth medium, with ground meat added, proved satisfactory for maintenance of cultures and toxin production. Although slight turbidity developed even at room temperature in air, heavy growth was not obtained under any conditions; and after a few days, cultures seemed to become clearer, suggesting some degree of lysis. Gas production was quite vigorous in the early stages of growth, but the meat was not apparently digested. Anaerobiosis had little effect upon growth in this medium; but when the ground meat was omitted therefrom, better growth occurred under anaerobic incubation. Addition of 2 per cent dextrose also markedly increased the rate and amount of growth, as evidenced by gas production, turbidity, and the formation of a whitish sediment. From the standpoint of toxin production, the effects of adding dextrose to the medium were somewhat unpredictable.

On well-dried human or sheep blood agar plates, incubated at 37°C. under complete anaerobiosis in a McIntosh-Fildes jar, small, flat, haemolytic colonies developed in 24 hours. Some of these colonies were very minute. After 72 hours of incubation, they reached a maximum diameter of 2-3 mm., and were translucent, faintly greyish, with a smooth or slightly irregular edge. Some plates showed a number of colonies with an indented or bitten margin, while other colonies showed glazed central or peripheral sectors. These phenomena suggested either bacteriophage activity or a special form of autolysis, but an attempt to isolate a bacteriophage from the cultures was unsuccessful.

On imperfectly dried blood agar plates, colonies were liable to assume irregular, branching forms, which developed arborescent, frond-like or spidery processes. If the plates were quite moist, this type of growth quickly became confluent, spreading as a thin film over wide areas, and even over the whole surface of the medium. In young cultures, the zone of haemolysis extended for 1-2 mm. beyond the edge of growth, its margin being diffuse, while decolorization of the blood was incomplete. Longer incubation often resulted in extensive and sharply demarcated haemolysis.

By incorporating the appropriate sugars in a semi-solid thioglycollate medium, with phenol red as indicator, the "VH" strain was found to produce acid and gas in the presence of dextrose, sucrose, maltose, levulose, and glycerol; whereas lactose, mannitol, dulcitol, xylose and arabinose were not fermented. These reactions appear characteristic of type E cultures (2). Numerous other sugars were tested, but gave somewhat inconstant reactions. Some discrepancies were noted in the effects of the "VH" and "Nanaimo" strains upon, for instance, galactose and salicin. Another point of difference between these 2 strains is that "Nanaimo" cultures had a putrefactive odour, while "VH" cultures were practically odourless, having only a faintly marine or seaweedy aroma.

In the ground-meat medium, or in peptone beef-infusion broth, toxin production by the "VH" strain reached a peak of around 4000 mouse M.L.D.'s per cc. after about 5 days' anaerobic incubation at 37°C. This figure represents a slightly higher toxigenic capacity than has been reported for other type E strains (4, 9, 2). Using mice as test animals, the toxin was shown to be unaffected by types A and B botulinus antitoxic sera. On the other hand, neutralization was readily demonstrated by means of an antitoxic serum prepared by immunizing a rabbit with "Nanaimo" type E toxoid.

A Rhode Island pullet, weighing 250 gm., developed paralysis of one leg and a wing, with

general weakness, after intramuscular injection of 500 mouse M.L.D.'s. In this respect, the "VH" strain differs again from the "Nanaimo" salmon strain, but resembles Hazen's salmon strain (10).

Possible Sources of Contamination

The chief alternative modes of access of *Cl. botulinum* to the herring will now be briefly considered. One possibility is that type E micro-organisms were introduced by the housewife at the time of bottling the herring in the pickling fluid, either through direct pollution from her fingers, or from the spices and chopped onions which were added to the vinegar. But the woman in question was intelligent and clean in her housekeeping habits, and was experienced in pickling. A sample of the actual spices used was examined bacteriologically. Many types of Gram-positive, sporulating bacilli grew in both aerobic and anaerobic culture; but none resembled *Cl. botulinum* in morphology or colonial characteristics. Moreover, though limited growth occurred when a commercial sample of pickled herring was experimentally inoculated with 1 cc. of a 5-day culture of the "VH" strain, the potency of the pickling fluid never exceeded 10-20 mouse M.L.D.'s per cc. over a period of several weeks at room temperature. Such an order of potency seems unlikely to have lethal potentialities for man in the given circumstances, and is in any event incompatible with the demonstrated presence in the victim's jejunal fluid of around 1000 mouse M.L.D.'s per cc.

The circumstances of the Vancouver episode seem more explicable by assuming that pollution with type E organisms, followed by their proliferation in the herring muscle, and their production of toxin in amounts lethal for man, occurred prior to the pickling. But this assumption in turn raises questions concerning the original source of these organisms, and their route of access to the fish muscle. In the Nanaimo fatalities (2), the postulated source was the soil of the garden or chicken run which a man had been digging just before carrying out a faulty home-canning process. Certain differences were subsequently noted between the type E strain isolated from the soil and the strains isolated from cans of salmon and chicken; but our reported findings nevertheless indicated that type E botulism could originate (as outbreaks of types A and B botulism often do) from direct pollution of a foodstuff with soil.

The chances of fish becoming polluted with *Cl. botulinum* from soil may at first seem rather slight, but on further consideration many possibilities can be envisaged. Sanitary precautions on these small boats are very crude; and from the time the herring are torn from entanglement in the gill nets until their ultimate consumption, they encounter numerous contamination hazards. They are, for instance, thrown into an unclean hold, heaped into old boxes or baskets, or left about the deck in the way of trampling boots. The integuments of the fish are thus soon liable to be exposed to bacteria, not only directly derived from soil, but also originating in slime, gut residues, fish faeces, and mud from the sea bottom. Since no ice is carried on board, the bacterial counts on the surface of the fish are already high by the time the boat arrives at the docks, where there is further exposure to trauma and pollution. Obviously, even if type E organisms normally subsist in a strictly terrestrial, as opposed to marine

habitat, then opportunities are not lacking for superficial contamination of herring therewith. Furthermore, although the intact skin of a fish presents a remarkably effective barrier to contaminants, any tear, cut, puncture, bruise, or other damage to its integrity may incite rapid penetration of bacteria into the underlying flesh.

An additional hypothesis merits some attention, viz. that in this and perhaps certain other fish-borne botulism outbreaks, the causal micro-organisms migrated through the gut wall into the adjacent muscle tissues. The fishing industry recognizes that enzymic digestion of the intestinal lining is apt to begin very soon after death, when the gut contains feed. Summer-caught herring are notoriously liable to rapid spoilage, especially when engorged with the so-called "red feed" plankton, a miniature shrimp-like crustacean which intermittently abounds in coastal waters hereabouts. At such times, the herring are very prone to develop "belly burn"—in which the flesh is stained from leakage of intestinal contents—and they are then considered unfit for human consumption. Mainly for these reasons, summer-caught herring are not suitable for commercial canning or pickling. A few may be kippered, but the majority have to pass muster for the local fresh fish market, or else go to the reduction plant for conversion into fertilizer. Realizing this, herring fishermen usually land their overnight catch at the docks early next morning, i.e. within 12 hours. But at times, 24 hours or longer may elapse between the catching and landing of these fish; and there is nothing to prevent anyone from going to the docks and bargaining for herring that may be damaged and stale.

Since there is apparently plenty of opportunity for invasion of herring muscle by bacteria migrating from the gut, the question arises whether *Cl. botulinum* type E could conceivably be sometimes numbered among such bacteria. The supposition that soil may be the natural habitat of these organisms is not inconsistent with this possibility. For the waters off Point Grey, one of the favoured areas for summer herring fishing, are heavily admixed with silt from the Fraser River and soil from mountain streams, as well as with sewage from the city of Vancouver and neighbouring agricultural districts. The possible significance of animal excreta as a source of *Cl. botulinum* in sewage-polluted waters should also not be overlooked. Easton and Meyer (22), in 1924, reported that hog manure quite frequently yielded toxic cultures of *Cl. botulinum*; and Meyer (23) later pointed out that several types of these organisms exist saprophytically in the intestines of certain animals. Later, Burov et al. (24) claimed that animals such as rats, which have a high degree of natural resistance to botulism, might intermittently excrete *Cl. botulinum* after experimentally ingesting the spores. Thus it would seem justifiable to contend that type E organisms might have been present in the local off-shore waters. Their absorption on to planktons floating upwards through waters polluted by soil and sewage, and hence their ingestion by herring feeding in the vicinity, could then follow fortuitously.

The hypothesis that lethal amounts of botulinus toxin were already present in the fish at the time of pickling, was strengthened by certain experimental findings, which will be reported in detail elsewhere. These experiments were designed to ascertain first, whether the time elapsing, and the environ-

mental conditions prevailing, were compatible with the production of toxin of sufficient potency to kill a man; and secondly, whether such preformed toxin would be significantly reduced in potency by exposure for several days to pickling fluid. In the fatal episode reported above, at least two consecutive days may be reckoned to have elapsed during which the fish were on board the boat, at the dock, and in the purchaser's house, before they were placed in the deep-freeze locker; and a few weeks later, there was yet another day during which they thawed out in the housewife's kitchen. In other words, the botulinogenic herring consumed by the two men had been exposed, unsalted, to summer temperatures for a total of not less than three days before being pickled. Our findings, summarized below, indicate that within this period, and at a similar temperature, fresh herring inoculated with the "VH" strain developed a degree of toxicity comparable to that found in the jejunal contents of the fatal case.

Two fresh herrings, one recently caught, and the other thawed out after storage in a deep-freeze locker, were inoculated intramuscularly with about 1 cc. of a young culture of the "VH" strain, and left in sterile beakers at room temperature (around 23°C.). Approximately 1-gm. samples were taken at intervals from each herring, macerated in physiological saline, and the mouse-killing power of the supernatant extracts determined. Within 72 hours, samples taken from different parts of the herring showed heavy growth of type E organisms, and the extracts developed a potency equivalent to at least 1000 mouse M.L.D.'s per gram of fish. (A salt herring, which had been kept in strong brine, did not support growth of the "VH" strain.)

Portions of the foregoing herring, of known high toxicity, were "pickled" according to the housewife's formula. These preparations were also kept at room temperature, and periodically subjected to bacterial counts and lethal potency tests. The pickling fluid exhibited a high, and to all intents undiminished toxicity, for several weeks. Meanwhile, the type E organisms only slowly declined in numbers, and indeed multiplied intermittently, despite the low pH range of 4.5-4.8. (A parallel experiment, in which the pickling fluid was saturated with sodium chloride, gave similar results.)

In another experiment, samples of macerated fresh herring were inoculated with the "VH" strain, and stored at various temperatures. Toxin production in the fish paste proceeded to higher potencies, and was maintained longer, at 23°C. than at 37°C. Even at 6°C., slight but definite toxin production was detectable.

Discussion

In this outbreak, as in the two preceding ones reported from British Columbia, the possibility of botulism was apparently not considered by the patients' physicians. Indeed, the true diagnosis might never have been established had the interne who admitted the patients to hospital not recollected a recent impressive lecture on food poisoning given by his professor of preventive medicine. The abrupt onset of an afebrile illness characterized by abdominal distress, general toxaemia, and paralyses of motor nerves (cranial, peripheral, or both), should suggest the possibility of botulism, and provoke enquiry into the nature of foodstuffs eaten within the preceding 2-3 days.

The cases described happen to illustrate certain variable aspects of the syndrome. Neurological phenomena were paramount in the fatal case, whereas the patient who recovered displayed mainly abdominal symptoms, with diarrhoea persisting throughout a prolonged convalescence. In the herring

outbreaks described by Ganelin and Billig (15) and by Wasmuth (12), diarrhoea and constipation respectively were conspicuous features in a number of cases.

Two comments on specific therapy seem pertinent. First, had a type E organism not been demonstrably responsible for these cases, the one patient's recovery might well have been ascribed, in part at least, to the administration of type A antitoxin. This situation resembles that reported by Mackenzie (6), in which a 15-year-old girl died, and her parents recovered, from botulism contracted as a result of eating smoked salmon appetizer at a luncheon-cocktail party. All of them received, intramuscularly or intravenously, a mixture of types A and B antitoxic sera. This was the salmon from which Hazen (10) subsequently isolated a type E strain of *Cl. botulinum*. Secondly, notwithstanding such pitfalls in deduction, and despite the dearth of evidence that administration of botulinus antitoxin of even homologous type may be of vital importance under the clinical circumstances liable to be encountered, it seems very unfortunate that no botulinus antitoxic serum of any type, let alone type E, is apparently available now in North America.

Too few isolations of type E strains have been made to warrant generalizations about their characteristics. Systematic study of a collection of these strains is now under way. For the present, besides their common toxigenic specificity, they can be said to exhibit the property of producing toxins of relatively low potency, in terms of mouse M.L.D.'s, when grown in the customary laboratory media; they also have a propensity for causing fish-borne outbreaks of human botulism, whose case fatality rate is nevertheless of the same order as in types A and B outbreaks. If it could be demonstrated that type E strains flourished and produced especially potent toxin in fish, both phenomena would thus be explained, and a search for any other special association between type E strains and fish would become superfluous. However, it might prove necessary to test alternative hypotheses, e.g. that type E toxin is perhaps somewhat more resistant than types A and B toxins, to the action of proteolytic enzymes encountered in the upper portion of the human gastrointestinal tract; or that type E toxin molecules are smaller, more quickly absorbed, and therefore less subject to proteolysis, than the molecules of types A and B toxins. These possibilities, considered alone, would not account for the apparent predilection of type E strains for fish.

This question of the affinity for fish of type E strains can also be viewed conjecturally from the standpoint of their natural distribution. On the one hand, these strains may be widespread in soils; but being less resistant to deleterious influences, or more fastidious in nutritive requirements, may seldom find conditions propitious for toxin production in the kinds of foodstuffs usually associated with types A and B botulism. On the other hand, perchance type E strains may be more apt to survive than types A and B strains in certain waters, salt or fresh. Kalmbach and Gunderson (25), and Quortrup and Holt (26), have demonstrated that some types of aquatic vegetation in "Western duck sickness" areas, serve as an excellent medium for *Cl. botulinum* type C growth and toxin production. Indeed, some analogy may eventually be traceable between the capacity of type C strains to thrive occasionally in the water and vege-

tation of stagnant alkali lakes, and the epidemiology of type E botulism in man. In this connection, we may fittingly mention here the mysterious deaths of large numbers of pilchards (27) and herring (28), reported in the coastal waters of British Columbia within the last 10 years. No satisfactory explanation of the mortality was forthcoming. The symptomatology and post-mortem findings were not inconsistent with a diagnosis of botulism, but unfortunately no anaerobic cultural work was done. Tides, currents, and an enormous dilution factor, quite apart from the chemical composition of sea water, render implausible any suggestion that a lethal concentration of type E toxin would accumulate, even in the relatively stagnant waters of some narrow inlet. But circumstances could conceivably arise wherein fish might ingest type E organisms, and also plankton feed—the latter furnishing a suitable medium for toxin production *in vivo*, with consequent death of the fish. All these speculations should eventually be resolvable by experimentation.

Finally, this episode may illustrate a serious risk inherent in pickled herring (and other forms of uncooked fish) which is insufficiently appreciated by persons of varied heritage, e.g. Jewish, Scandinavian, who enjoy such food. Even in the most careful hands, fish are exposed to numerous opportunities for bacterial contamination. Their reputation for being seldom implicated in any kind of bacterial food poisoning is perhaps due more to the rapidity with which fish undergo spoilage than to the effectiveness of any special hygienic precautions. In these circumstances, it would seem foolhardy to rely upon vinegar—at best a rather poor bacteriostatic agent—as the sole means of preserving uncooked fish. These 2 cases of botulism also exemplify the futility of assuming that foodstuffs which have been stored in a deep-freeze locker are necessarily safe. In fact, in this particular instance, the low temperature at which the fish were held for some weeks prior to being cleaned and pickled, presumably spared preformed toxin from degeneration; and by allowing the type E organisms or their spores to survive, made further proliferation and toxin production feasible while the fish were thawing out.

Summary

1. One man died, and another recovered, after consumption of home-pickled herring caught near Vancouver, B.C. From the stomach and jejunal contents of the fatal case, and from a fragment of discarded herring bone, *Clostridium botulinum* type E was isolated.

2. The total number of outbreaks of botulism recorded in Canada now stands at 6. In all, 38 persons have been affected, 22 fatally. The causal micro-organism was identified in 3 of these outbreaks, all occurring in British Columbia within the 5-year period 1944-49, and involving 7 persons, of whom 6 died. Two of the 3 outbreaks recorded in this Province have been ascribed to type E organisms, with fish implicated as the vehicle.

3. The "Nanaimo salmon" and "Vancouver herring" strains are alike in many respects, but differ in certain particulars. For example, whereas toxin from the Nanaimo strain does not affect chickens, an equivalent dose of "VH" toxin (as judged by mouse M.L.D.'s) caused paralysis on intramuscular injection into a pullet.

4. Only 5 type E outbreaks of botulism have been recorded to date, all of them in North America. Among the 14 persons affected, there were 7 deaths. The prototype E strains were originally isolated from fish in the U.S.S.R., but were identified in the United States as representatives of a new type.

5. The high predilection for fish shown by type E strains is the more conspicuous because in the United States botulism has seldom been traced to fish. In Russia, and to a lesser extent in Germany, fish-borne botulism seems to have been relatively common; but little bacteriological information is available respecting types involved there.

6. Consideration has been given to the routes and mechanisms whereby fish may be polluted with *Cl. botulinum* and hence become botulinogenic to the human consumer. In the particular episode described, the toxin was almost certainly elaborated in the flesh of the herring prior to the pickling process. The type E organisms may have reached the muscle tissues from damaged and contaminated integuments, or by leakage from the gut.

7. Brief reference is made to many potentially botulinogenic hazards inherent in current methods of handling fish. Pickled herring would seem to be especially vulnerable from this standpoint.

Acknowledgements

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CORRECTION

Reference 3, page 132, volume 41 (March, 1950) should read:

Felix, A. and Callow, Bessie R., Brit. M. J., 1943, July 31, vol. II: 127.

Cancer Mortality Trends in Different Countries

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AN examination of cancer mortality trends in the Canadian provinces has failed to show any differences between provinces in spite of vast differences in "control" measures. It has also failed to show any decline in recorded mortality from either breast cancer or all cancer or any change in trends that could be credibly attributed to increase in "control" measures over the past twenty to twenty-five years. During that period there has been widespread propaganda stressing the value of and urging early treatment; as far as can be ascertained, there has been very considerable increase and acceleration of treatment in some provinces; in some provinces heavy expenditures have been made in setting up and maintaining elaborate diagnostic and treatment organizations in a very aggressive effort to "control" cancer mortality; in that period there have been many advances in medical practice in general and other reputed advances more specifically related to the diagnosis and treatment of cancer, including practically all the development and application of radiation therapy beyond its foundation; and besides these factors there have been many others generally considered favourable to a reduction in cancer mortality, such as improved transportation, greater use of specialist services, a more ready acceptance of extensive treatment, improved economic situation, vastly greater hospitalization, etc. The uniform lack of recorded decline in all the provinces regardless of programs, facilities and services, with the bookkeeping for all the provinces for all mortality done by the Dominion Bureau of Statistics, shows as clearly as vital statistics could show the failure in Canada of organized "control" measures and of all the other factors supposedly operating to reduce cancer mortality (1, 2, 3). Thus, a comparison of the Canadian experience with that of other places is indicated. For this purpose, the rates for England and Wales and for Massachusetts are available.

The Data and Their Sources

The rates for England and Wales have been calculated from data given annually in the Registrar-General's Statistical Review (4). Unfortunately, there is a serious break in continuity of the data subsequent to 1939. This is due, in part at least, to two factors introduced in 1940 and well recognized. One of these, changes in classification, had, as shown in the Review for that year, very little effect on the rates in the age groups under consideration here. However, the second factor, a drastic change in the selection of primary cause

to which the death is charged, had a very marked and immediate effect on some of the cancer rates. As is pointed out in the Review, the effect varied greatly in different age groups. There is reason to think, too, that it will vary somewhat in time as the medical profession realize more fully their responsibility in regard to certification and modify practice accordingly. The operation of another factor or factors is very strongly suggested, if not frankly revealed, in an unusual, marked decline in the rates for all causes in older age groups in this period. At variance with previous experience and with what might be expected in wartime, and at variance, too, with the experience of the same time in Canada, this decline has all the appearance of artificiality. There has been no census since 1931 and population estimates of 1940 onward have been based on the National Register enumeration of 1939; as the census in 1921 revealed gross discrepancies in the pre-censal estimates of some age groups at that war and post-war period, it would not be surprising if similar discrepancies again crept into the estimates of this later war and post-war period, even farther away as it is from a census enumeration; thus, population estimates might be one of several possible factors apparently distorting the rates for all causes. Whatever the factor or combination of factors might be, it would operate also in cancer mortality records, distorting their comparability. With the data at hand, correction cannot be made satisfactorily in the different age groups for the change in selection of the cause of death, nor can correction be made for any factor or factors suspect but not definitely identified. As it would be misleading to compare the patently incomparable data from 1940 on with previous data or with other data and as adjustment to comparability cannot be made, these data (1940 on) must be eliminated from the comparison here. This does not, it is thought, seriously interfere with wider comparison as it is extremely unlikely that the war or post-war conditions would yield any marked improvement in cancer mortality over that of the previous period. Even the data for the years approaching and including 1939, at some distance from a census enumeration, carry more than the usual degree of uncertainty, but as adjustment of them to the 1939 National Register enumeration would not, it is feared, ensure greater comparability, that adjustment has not been made.

The Massachusetts rates have been graciously and generously provided by Dr. Herbert L. Lombard, Director of the Division of Cancer and Other Chronic Diseases, Department of Public Health, Massachusetts. For many reasons it is particularly fortunate to have the rates for this State. Massachusetts has one of the oldest and, by common consent, one of the best organized "control" programs in the world, one engrafted in 1926 on routine medical and public health services of, again by common consent, the highest order, their practice reflecting for over 100 years an alert social consciousness on the part of the profession and the people. Even before the turn of the century, concerted action regarding cancer was taken, and for decades, Boston, with its great medical schools, hospitals and clinics, has been recognized as one of the leading medical centres of the world. Something of the force of the "control" program and of the degree of acceleration of treatment since 1926 is evident in Dublin's (5) summary statement that the "Massachusetts data show that the

average time elapsed between first symptom and visit to the physician dropped from 6½ months in 1927 to a little more than three months in 1945; the average time elapsed between first visit to the physician and visit to the clinic decreased from 5½ months in 1927 to less than three months in 1945. The proportion seeking medical consultation within one month of first symptoms increased by more than half."

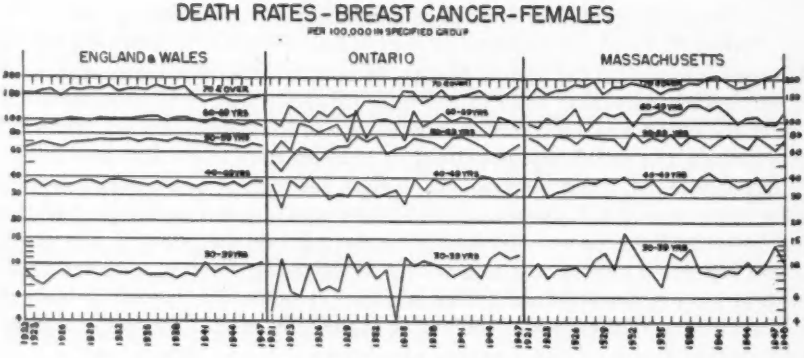
The data for each of the Canadian provinces, compiled and tabulated since 1921 by the Dominion Bureau of Statistics, are available in the annual reports of the Registrar-General of the respective province and in the annual VITAL STATISTICS, CANADA, published by the Bureau.

THE TRENDS IN THE RATES

Breast Cancer

It is apparent in Graph I that the breast cancer mortality rates for England and Wales are of approximately the same height as the Ontario rates and that the Massachusetts rates are slightly higher. This difference may be, and probably is, one of diagnosis, certification and/or book-keeping; and, as it might be reasonably assumed that not only the occurrence of breast cancer but also the diagnosis and certification in Massachusetts would be closely similar to that in Ontario while the book-keeping is under different auspices, it is probable that this factor, the book-keeping, accounts for most of the difference. Although the relative height or magnitude of the rates is not under consideration in this note, the difference serves to demonstrate how rates may vary from place to place, and how, in comparison of such rates, the causal factors of such variations must be determined. But it is the trends of the rates rather than their magnitude with which this note is concerned.

The Ontario rates for breast cancer mortality, analysed in more detail previously, show some irregular increases in the early 1920's and more prolonged increases in older life. The earlier increases have been attributed to two factors with which they coincide—the adaptation of the Dominion Bureau of Statistics to its new responsibility, the book-keeping of vital statistics for



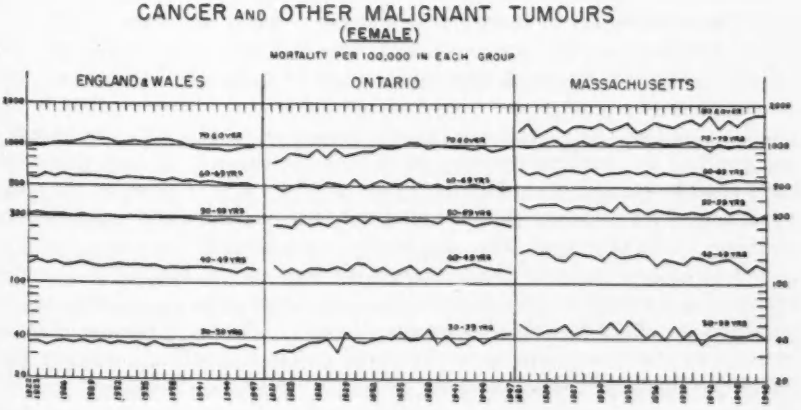
GRAPH I.

the provinces, and, too, the return to civilian practice of a large proportion of the medical profession, possibly with some quickened regard or facility for records and precision. The more prolonged increases in older age have, too, been credibly attributed to progressively greater precision in diagnosis, certification and book-keeping of these deaths; what was charged previously to senility is now charged to more specific causes. But the most striking and most significant feature of these Ontario rates is the maintenance of a near level for the past fifteen to twenty years in each age group without any suggestion of any continuous decline.

The English rates (to 1939) show much the same picture but, based on larger figures, give smoother lines. There is a slight increase in older age similar to but smaller than that in Ontario. Otherwise, the English rates, like the Ontario rates, fairly maintain their respective levels. So, too, the Massachusetts rates for breast cancer mortality practically reproduce, though at higher levels, the trends in Ontario; they show some increase in older age with the maintenance of levels in the other age groups. The Massachusetts rates and the Ontario rates, on account of the smaller numbers, show much irregularity with wide and prolonged fluctuations.

All Cancer

The rates for all cancer (females) in Ontario show, as in breast cancer, slight increases in the 1920's, more prolonged in the 70 and over age groups; they then hold close to a level for the rest of the experience. The English rates (to 1939), on the other hand, show appreciable but varying declines in all age groups under 70. The decline is perhaps greatest in the 60-69 age group where the onset, though very indefinitely marked, appears to date back to the 1920's, since when the trend has been regularly downward with but little fluctuation. In the 50-59 age group, the decline is even more indefinite in its onset, and indefinite, too, in its extent and its course; while there is a per-



GRAPH II.

ceptible fall in the 1920's, the line holds very close to a level for the next decade. In the 40-49 age group and in the 30-39 age group, the decline is similarly uncertain in onset, course and even extent, but, over the years, is appreciable. The Massachusetts rates, at distinctly higher levels than the Ontario rates, naturally show, like the latter, more fluctuations than the English rates based on larger numbers. In the 80 and over age group, there is an increase comparable with that in old age in Ontario. In the 70-79 age group, the rates maintain a fair level. In the 60-69, 50-59, 40-49 and 30-39 age groups, they exhibit some declines, but the declines vary in time and type of onset, in course and in extent. In the 60-69 age group, the decline appears to be delayed until after 1940; it is then abrupt and carries the rates down in two years from a level of over 600 to about 550, near which the rates have since been maintained. In the 50-59 age group, the fluctuations make it difficult to identify the onset or to trace the course of the decline. In the 40-49 age group, the rates seem to hold near a level of over 200 until about 1935, then a slightly lower level until 1945 when they drop to 140-150 for the last three years. Again, the onset cannot be identified or the course traced with any assurance, nor can the extent be profitably measured. The rates in the 30-39 age group, with even wider fluctuations, suggest a course somewhat resembling that in the 40-49 age group. Thus, in summary, while the Ontario rates for all cancer mortality do not show any assured decline in any age group, the English and Massachusetts rates exhibit declines, small but appreciable and varying in time and type of onset, in course and extent. In some, the decline appears to date back to the 1920's or earlier; in others, it is delayed until much later. In some, the onset is practically imperceptible, in others, it is well marked; in some, the course is regular and steadily downward; in others, it either gradually or abruptly carries the rates down to a new level at which they are then maintained.

The Significance of the Levels and the Recorded Declines

The maintenance of the respective levels in recorded breast cancer mortality in all the Canadian provinces, regardless of "control" measures, and also in England and Wales and Massachusetts, cannot be lightly disregarded. The maintenance of a level in any rates is less apt to be due to artifact than is any change, small or large, abrupt or gradual. As the records of any cause of death are much more susceptible to change than is the cause itself, any change in rates must be suspect of artifact until proven otherwise. A level, on the other hand, suggests a uniform relationship of the rates to reality and suggests, too, stability of the problem. The maintenance of a level in the rates during a period of intense publicity and interest devoted to the subject is stronger evidence of the reality of the rates and of their relative insusceptibility to the influence of publicity or other extraneous factors. The maintenance of levels in the rates of different provinces with vastly different "control" measures, but under one auspices for book-keeping, practically confirms the general validity of the rates, confirms, too, the lack of any real decline in mortality and thus clearly establishes the failure of "control" measures. The maintenance of

levels in the rates of three different countries under different book-keeping auspices adds another strong link in a strong chain of consistent evidence of the practical invulnerability of breast cancer mortality to present-day measures of so-called "control".

The failure to reduce mortality from breast cancer, as evidenced by the recorded rates, is quite in keeping with but enlarges on the inference to be drawn from the statement of Nathanson and Welch of the Cancer Commission of Harvard University who, in 1936, reported a comparison of hospital data for two periods (6): "There is no absolute proof from these data," the authors say, "that the treatment of carcinoma of the breast has increased the life expectancy significantly in the years 1923-1932 compared with that of the years 1912-1922. The Huntington group shows a definite improvement in the second decade, but this is probably due to a more favourable group of cases."

The lack of any decline is in conformity, too, with the doubts of Halsted (7) and of Lewis (8), both of whom recognized clearly the incompleteness of and possible fallacy in the currently accepted evidence of the high efficacy of early and radical treatment of breast cancer; they recognized great gaps in our knowledge and understanding of the natural course of "cancer", and Lewis, particularly, in 1932, pointed clearly to deficiencies and probable fallacies in the accepted diagnostic criteria and indicated this field as one in need of investigation. Time has amply vindicated their caution and verified their foresight. Sir Gordon Gordon-Taylor (9), too, "a simple soul," he says himself, who has "never sat among the mighty in Cancer Campaigns" but who reports an unexcelled ratio (80-85 per cent) of cures by early treatment, nevertheless, has forcibly emphasized great deficiencies in our knowledge and understanding of metastatic development—deficiencies which affect our judgments and appraisals of treatment. Further, the lack of decline in breast cancer mortality, as shown in mortality rates, adds little to Truscott's conclusion from a survey of Middlesex hospital cases, previously quoted but worth repeating, that "no matter how early the case or thorough the treatment, no patient is free from the possibility of recurrence until death occurs from some other cause" (10).

With such evidence of failure in "control" of breast cancer—of all major cancers the one most readily accessible for diagnosis and treatment; the one given, by the records of many clinics, the highest probability of cure if treated early; the one accorded the most emphasis in propaganda; the one in which there has been the greatest response to propaganda and the most determined effort to "control"; the one with the least play of change in diagnosis (at death), in certification and/or book-keeping in the past twenty years—with such consistently strong evidence of failure to "control" breast cancer mortality, evidence of unimpeachable quality should be required to carry conviction of reality in any recorded decline in mortality from any other major cancer, less accessible for diagnosis and treatment and much more susceptible to change in the records.

As noted, however, the records show declines in the mortality of all cancer in England and Wales (to 1939) and in Massachusetts but not in Ontario or Province B or in the other provinces of Canada.

These declines, if real, would indicate greater cancer "control" in England and Wales and in Massachusetts in the past twenty to twenty-five years than in any of the Canadian provinces, due to superiority of some factor or factors —more effectual propaganda, better response of the people, provision of more adequate diagnostic and treatment facilities or a medical profession of higher calibre able to take greater advantage of an earlier demand for attention. Obviously, no fine comparisons can be drawn. But it is to be noted that propaganda in both Ontario and Province B has been widespread and persistent; official, semi-official, voluntary and commercial health agencies (federal, provincial and municipal) have all played a full part, and university staff members, physicists, pathologists, radiologists, surgeons and physicians have participated generously, giving freely of themselves and their time, with their words, both in print and on the air, carrying strong persuasion from the force of their personality, the weight of their experience and the prestige of their position. The medical press and the public press and the radio have given constant publicity to the advantages offered by earlier diagnosis and treatment. In both provinces, local organizations have been formed in many communities for the purpose of interesting and "educating" the public. Literature has been supplied by the most promising routes to the most promising subjects. The "educational" program has been carried so far that school children of fifteen and sixteen, writing their examination on the subject of Health(!), have been confronted with the questions: "State two symptoms of cancer," "State the two commonest causes of cancer," and "State two recognized treatments of cancer." It is difficult to contemplate anything more widespread, more persistent, more pressing or more personal in its appeal. There is evidence, too, of an increase and acceleration in treatment due to propaganda and other factors. In Province B, the clinic attendance has so increased, with no cancer found in over half of those now presenting themselves, that action has been taken to curb the attendance. In Ontario, at one clinic there are 1,000 women on the waiting list for examination to have cancer diagnosed or "to be assured that there is no disease lurking in the body." At private clinics in both provinces, women in dread of cancer but without symptoms have presented themselves in embarrassing proportions (11). It would thus appear that the response of women in these two provinces has been, as far as numbers are concerned, all that could be expected or desired. Nor have services and equipment been wanting. Both provinces have provided pathological services for cancer diagnosis. Both have developed extensive radon emanation plants. In Province B, a betatron unit was installed in 1949, and, in Ontario, a radioactive "cobalt bomb" has been supplied this year to one of the clinics. Province B, in 1944, provided for full diagnosis and treatment of all cancer without any direct charge to any patient. In Ontario, clinics readily accessible to all the population have been organized and operated. These highlights, though only highlights, indicate that the provision of equipment, facilities and services for the diagnosis and treatment of cancer in these provinces has not been on any stinted scale but rather the reverse. Hence, it is extremely doubtful that the propaganda in England and Wales and Massachusetts has been more persuasive or that there has been greater

response on the part of the people or that the provision of facilities and services has been any more lavish. To even imply comparison of professional calibre is odious. Certainly the uniformity of the level trends of breast cancer mortality in all three countries does not suggest any superiority or inferiority, qualitative or quantitative, of the medical attention, nor does the complete uniformity of the trends in both breast and all cancer in the different provinces of Canada give any such indication. Thus, it is difficult to identify any factor to which the difference in trends in the different countries can be charged.

Are the declines, then, real or not real? No categorical answer can be made to this question. Nevertheless, their features give some indication of their reality or unreality—an indication as free as possible from emotional or other bias. The declines, as noted, are dissimilar and inconsistent in different age groups. Some suggest a concentration of effort in time and in age entirely at variance with reality. The onset varies in both time and character; in some, it is almost imperceptible and dates back to the 1920's or earlier; in others, it is abrupt, varying in time in different age groups but carrying the rates down in one or two years to a lower level where, in some cases, they appear, then, to be maintained. Not one of the recorded declines shows such an accelerating rate of decline as would be consistent with the nature of cancer and the application of "control" measures. Thus, the declines exhibit few or none of the attributes suggestive of reality—reasonable consistency in extent in different age groups (or, perhaps, greater in the 40's and 50's than in older life), consistency in course, with, in all, an accelerating rate of decline. On the other hand, their inconsistencies and uncertainties are entirely compatible with the operation of artificial factors.

This interpretation of the rates is in agreement with the terse and cautionary statement of Ralston Paterson (12), Manchester, England, who, in 1942, in discussing the Cancer Act and proposing improvements for administration, pointed out that "nothing medicine has done so far has had the least visible effect on cancer death-rates. . . . If we found that we could alter the vital statistics in these diseases, we would know that the methods had been successful."

At best, these declines are not the unimpeachable evidence required for conviction of reality of decline in other cancer mortality in the face of much superior evidence of no real decline in that of breast cancer; nor are they of a character to warrant any relaxation or modification of conclusions drawn previously from the Canadian data—that the nature of the neoplasm rather than the time and type of treatment is the important determinant of the final outcome and that metastasizing cancer is, in general, incurable in spite of early treatment.

Whether or not the recorded declines are real or any part of them real, or any expansion of them that might reasonably be made to allow for changes in diagnosis, etc., whether or not they actually represent small declines in certain cancers or a proportionally larger decline in some one cancer, by chance obscured in the Canadian rates, they are not commensurate with the large reduction promised in propaganda, or with the high cures claimed by many clinics, claims from which the propaganda stems, derives all its persuasive

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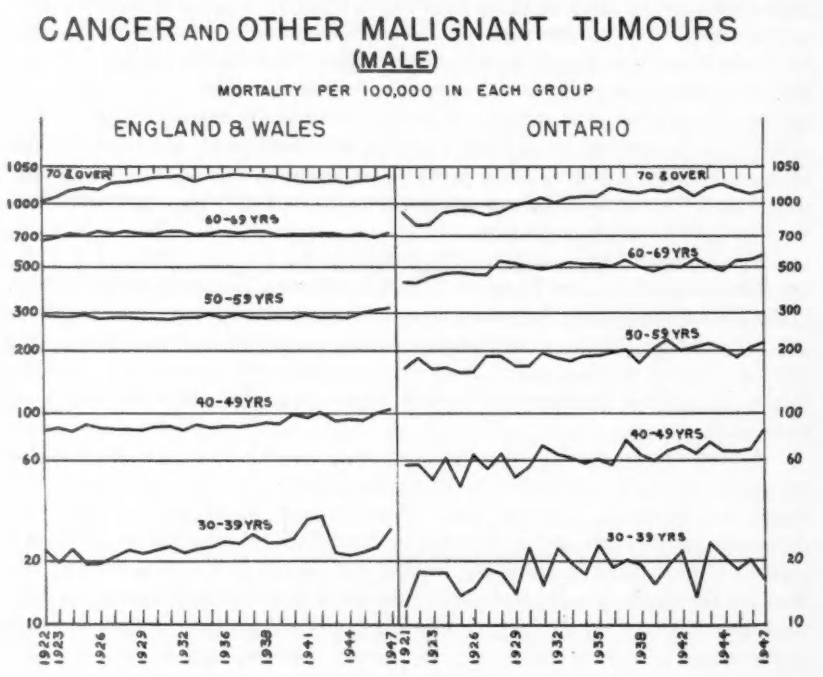
force and is sustained; they do not support the general concepts* of our ability to "control" cancer which have led to very heavy expenditures of public moneys for that purpose; they are greatly at variance with the general impressions* of achievements in that regard in the past two or three decades.

As the rates for cancer mortality in males have the further complexity of a much larger proportion of non-accessible cancer than the female and, therefore, much greater play, over the past 20-25 years, of change in diagnosis, etc. distorting comparability, and as they show no declines either in England and Wales or Ontario (Massachusetts rates are not at hand), discussion of them can be dispensed with here; moreover, the Ontario male rates have been considered in detail previously. Graph III, showing the England and Wales rates in each age group at much higher levels than the Ontario rates, is presented purely as a matter of interest; the reason for the difference in these rates is not for consideration in this note but it poses another problem for exploration.

Skin cancers—or what are called skin cancers—have never, in Canada,



GRAPH III.

*"Of course, we're curing lots of them; we wouldn't be doing them if we weren't." This reply to a question regarding the results of treatment of breast cancer typifies the belief of the majority of those responsible for doing treatment—a settled assurance of a high capacity to cure and equal assurance that a great many and a large proportion of cases are being cured. That belief is not restricted to breast cancer but is held similarly for uterine, cervical and some other cancers as well. A minority—some of them already quoted—have arrived at different conclusions from their experience.

contributed more than a small fraction of cancer mortality. The Egyptians, so the story goes, knew well the value of arsenic in the treatment of these lesions. There have been undoubted improvements in and more general acceptance of treatment so that the extensive destructive effects and deaths from rodent ulcer are even more seldom seen now than formerly. But the ability to cure such lesions, only locally malignant, does not indicate an equal degree of ability to cure metastasizing cancer and the two are not to be confused in any consideration of cancer or in any evaluation of "control".

While, for treatment, anything that can be done must be done, it should be done with full realization of the present limitations of diagnosis as well as of treatment.

Summary

The trends of breast cancer mortality and all cancer mortality, by age and sex, in England and Wales and Massachusetts in the past twenty to twenty-five years are compared with the trends in Ontario, the last being fairly representative of all the Canadian provinces.

Owing to the incomparability of the England and Wales rates subsequent to 1939, comparison has had to be limited, for them, to the previous period, 1922-1939.

Within that limitation, breast cancer mortality shows no decline in any age group in either Ontario (or any province of Canada) or England and Wales or Massachusetts. The rates maintain their respective levels.

The maintenance of levels of breast cancer mortality in three different countries, under three different book-keeping auspices, is very strong confirmatory evidence of the lack of any real decline in breast cancer mortality in either Canada, England and Wales or Massachusetts. The lack of decline in any age group indicates clearly the failure of "control" measures.

The all-cancer mortality rates in Ontario (and other Canadian provinces) show no decline in any age group but both the England and Wales rates (to 1939) and the Massachusetts rates show declines in practically all age groups under old age.

The declines, however, are not found explicable on the basis of more persuasive or wider spread propaganda, or greater response of the people to the propaganda, or more adequate provision of facilities and services or on a higher professional calibre. In their variability in extent and course from age group to age group and in time and type of onset, and, too, in the lack of any consistent acceleration in the rate of decline, they suggest paper changes rather than real declines.

But real or artificial, or to any extent real, or expanded reasonably to allow for vagaries in vital statistics and whether or not representing declines in some cancers or a proportionately larger decline in some one cancer, the recorded declines are patently incompatible with the optimistic promises of propaganda and with the glowing claims of cure of many clinics—claims from which propaganda stems, derives its force and is sustained; they are not compatible with the general concept of present-day capacity to "control" cancer mortality or with the general impression of achievements in that regard

in the past two to three decades. They do not justify the heavy expenditures that have been made with the objective of "controlling" cancer except in so far as the failure to achieve effectual "control" broadens our understanding of cancer and demonstrates that "getting cancer early" does not effectually "control" mortality. The ability to cure locally malignant lesions is not to be misinterpreted as an indication of equal ability to cure metastasizing cancer.

Treatment must be given as required but with full realization of its limitations and of the limitations, too, of early diagnosis and of our understanding of metastatic development.

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Control of Pollution by Federal Authorities

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SINCE the Department of National Health and Welfare is primarily concerned with the health of Canadian citizens and the Public Health Engineering Division is responsible for the application of engineering principles for the purpose of preventing disease, emphasis will be placed on the health aspects of pollution control by Federal authorities.

The importance of water to all living things and to the economy of our country is taken for granted. So much so that sometimes it is necessary to pause to consider it for a moment so that its place in our lives may be better appreciated. It has been the subject of poetry and song. Consider the following verse from a poem by Walter de la Mare:

Very old are the brooks;
And the rills that rise
When snow sleeps cold beneath
The azure skies
Sing such a history
Of come and gone,
Their every drop is as wise
As Solomon.

Suppose that one drop of water could be traced from the dawn of creation to now, and suppose it could sing a song of its own history as the poet suggests. How much of the earth would be unknown to it, if, during its existence, it was free to move from place to place, governed only by the laws of nature—a drop of rain, a brook or lake, a vapour attracted by the sun, a drifting cloud.

Probably on many occasions it was part of a body of water which was polluted or contaminated. What is meant by these terms? Our answer would be influenced by our particular interests. In "Preventive Medicine and Hygiene" by Rosenau, the following definitions are given:

"A *polluted water* is one which has suffered impairment of physical qualities through the addition of substances causing turbidity, color, odor or taste."

"*A contaminated water* is one which carries potential infection by reason of the addition of human or animal wastes or which has been rendered unwholesome by poisonous chemical compounds."

The word pollution, as commonly used, embraces both these definitions.

Presented at the annual convention of the Ontario Federation of Anglers and Hunters, held in Sudbury, Ontario, January 31, 1950.

In a recent pamphlet, issued by the Atlantic Salmon Association, entitled "Pollution Legislation in Canada" by Percy E. Nobbs, the author states that men and gulls are the chief defilers of water. He further states that, if the effects of industrial wastes are considered, man is the dirtiest animal in creation.

That may be too severe an estimate of our place in the animal kingdom but there is enough truth in it to cause serious thought.

A few fallacies have undoubtedly had much to do with the abuse of streams and lakes. One of these is the thought, almost belief, that water which is clear is safe, or, what you cannot see will not hurt you. Another is the widespread belief in the purification value of rapids, falls, and sand or gravel deposits on lake and river beds. Mixing and aeration of water are extremely important in the processes of purification but have not the cure-all values attributed to them by many people. Such fallacies seem to have been used to justify our practice of polluting surface waters. How else can we explain the practice of discharging sewage into our neighbor's water supply? How else can we account for the fact that so many of our municipalities discharge untreated sewage near their own water intakes? With this in mind, it is not too difficult to justify the statement that man is the dirtiest animal in creation.

What is being done about it? I believe it can be truthfully stated that more thought is being given to the problem of reducing pollution in North American waters than ever before. More people are conscious of the need for improvement and this is most important since treatment works are costly and funds must be provided by the taxpayer.

But what is being done by Federal authorities? In order to answer that question it is necessary to review, very briefly, the legislative authority under which action might be taken to bring about improvements. Such legislation is to be found in the British North America Act or in subsequent acts and regulations based on the authority allocated to the Federal Government and the Provincial Governments by the British North America Act. Section 92, paragraphs 10, 13 and 16, of that Act states:

"In each province the legislature may *exclusively* make laws in relation to matters coming within the classes of subjects hereinafter enumerated, that is to say,—

10. Local Works and Undertakings other than such as are of the following classes:
   (*a*) Lines of Steam or other Ships, Railways, Canals, Telegraphs, and other Works and Undertakings connecting the Province with any other or others of the Provinces, or extending beyond the Limits of the Province:
   (*b*) Lines of Steam Ships between the Province and any British or Foreign Country:
   (*c*) Such Works as, although wholly situate within the Province, are before or after their Execution declared by the Parliament of Canada to be for the general Advantage of Canada or for the Advantage of Two or more of the Provinces.
13. Property and Civil Rights in the Province.
16. Generally all Matters of a merely local or private Nature in the Province."

The introduction to Section 92 is repeated.

"In each province the legislature may exclusively make laws in relation to matters coming within the classes of subjects hereinafter enumerated."

Your attention is called to the word "exclusively". It is apparent that there is no possibility of Federal action in regard to controlling the discharge of municipal wastes. Those wastes contain most of the industrial wastes.

In some other phases of the pollution problem, the Federal Government has wider powers.

For example, the Navigable Waters Protection Act gives the Federal Government authority to protect the interests of navigation in such waters. Since any stream or lake which will float a log is navigable water, it is apparent that this act applies essentially to all our waters. You are probably aware that a Member of Parliament from Ontario introduced, during the recent session, a private bill to extend the authority of the Navigable Waters Protection Act to include pollution. In commenting on this bill the Minister of Transport is reported as saying to the House of Parliament: "The act does not deal with the pollution of navigable waters; it is concerned with navigation and the protection of navigation." Parliament decided that the proposed legislation could not be enacted under the authority of the Navigable Waters Protection Act and the bill was defeated. Pollution which could interfere with shipping might be controlled under this Act, but not the kinds of pollution in which we are interested.

Again there is the Fisheries Act. It, in turn, contains sections which deal with pollution. Section 33 sets out in considerable detail the substances which may not be discharged into fishing waters. No direct reference is made to sewage or industrial wastes although possibly both might be classed as "other deleterious substances". It is well known that gross pollution may so reduce the oxygen content of water that fish cannot live. Also certain wastes, such as cyanides, are very poisonous and have caused extensive fish kills in recent years in the Great Lakes and connecting waters. Other wastes, particularly sawdust, cover spawning beds and seriously affect the proper maintenance of the fish population. The Federal Department of Fisheries enforces this act in Prince Edward Island, Nova Scotia, New Brunswick and British Columbia with its own officers, but the other provinces are responsible for the enforcement of regulations established under the authority of the Fisheries Act.

Other Federal legislation with which you are probably all familiar is The Migratory Birds Convention Act. This is enforced by the Dominion Wildlife Service branch of the Department of Resources and Development, with the Royal Canadian Mounted Police as enforcement officers. It is of particular interest because in Section 56 of the Regulations established under the authority of this act, it too prohibits certain forms of pollution which may be harmful to wildfowl, specifically oil. Several occurrences of oil discharge which have destroyed considerable numbers of wild ducks have received wide publicity in recent years. Where such occurrences are the result of negligence or carelessness, the responsible parties can be prosecuted under this act.

Section 5 of the Department of National Health and Welfare Act outlines the duties, powers and functions of the Minister as follows:

"The duties, powers and functions of the Minister shall extend to and include all matters relating to the promotion or preservation of health, social security and social welfare of the people of Canada over which the Parliament of Canada has jurisdiction, and, without restricting the generality of the foregoing, particularly the following matters". Six subsections follow which deal more specifically with certain phases of the Department's responsibility but no specific reference is made to the control of pollution. Sub-section (f) reads as follows:—"the enforcement of any rules or regulations made by the International Joint Commission, promulgated pursuant to the treaty between the United States of America

and His Majesty relating to boundary waters and questions arising between the United States of America and Canada, so far as the same relate to public health."

As the International Joint Commission has no such rules or regulations, there is nothing to enforce at present. However, it might be worthwhile to review some of the activities of that commission during the past few years to indicate what action has been taken to improve the quality of the boundary waters between Canada and the United States.

As a result of complaints concerning the quality of waters of River St. Clair, Lake St. Clair and the Detroit River, the two countries concerned submitted a joint reference under date of April 1, 1946, to the International Joint Commission. Without reading the terms of reference in detail, they might be briefly summarized as follows:

The commission was instructed to ascertain the amount and character of the pollution in the waters concerned to determine whether pollution crosses the international boundary, to the injury of health and property on the other side of the boundary, and if so, to recommend what should be done to correct conditions. The resulting investigation was started in 1946. The terms of reference were extended to include the St. Marys River under date of October 2, 1946. Similarly an investigation of the Niagara River was authorized when the same reference was again extended under date of April 6, 1948.

Boards of Technical Advisers were established by the Commission to direct the investigations. For the investigation of the St. Marys River, St. Clair River, Lake St. Clair and Detroit River, the Board consisted of two representatives from each of the following: The United States Public Health Service, representing the United States Government, the State of Michigan, the Province of Ontario, and the Department of National Health and Welfare representing the Federal Government. Most of the field work connected with this investigation was completed in about two years and the Board submitted its report to the International Joint Commission in October 1949. This report has been accepted by the Commission but will not be available for distribution until the Commission completes its own report to the two governments. Speculation concerning the recommendations which will be made to the two Federal Governments would be inadvisable at this time. However, those who have been connected with the investigation are well aware of worthwhile improvements that have already resulted and feel confident that much more will be done to remedy undesirable conditions. This was one of the most comprehensive investigations in recent years. To give you an idea of the amount of work involved in the investigation, the following figures may be of interest. Sixty-seven industries were studied; 11,933 samples of water were analyzed for bacterial content, and 14,279 samples were collected for chemical analyses from which 73,480 determinations were made.

The Board of Technical Advisers for the Niagara River investigation was much the same as for the areas already mentioned, except that the State of New York had two representatives to take the place of those from Michigan. Field work in the Niagara area was completed in about one year and the Board of Technical Advisers is now preparing its final report.

The Commission has other investigations in progress in other parts of

Canada. Pollution in the Fraser River, B.C., is being studied, although power development is regarded as the most important phase of that investigation.

At the request of the International Joint Commission, the Public Health Engineering Division of the Department of National Health and Welfare carried out a short investigation of the Milk River in Alberta. This stream rises in the United States, flows into Canada and then crosses the boundary into the United States again.

In all this work of the International Joint Commission, the Federal Government is represented by its professional employees who are specially qualified for the particular investigation. Reports are summitted to the Commission which reviews them and usually submits a supplementary report to the Governments of the United States and Canada. The two governments then decide what action shall be taken to enforce the recommendations of the Commission. It is the proud claim of the Commission that in its existence of about forty years only once have the governments failed to endorse the Commission's findings and recommendations.

There are other phases of pollution control in which the Federal Government, and particularly the Department of National Health and Welfare, is vitally concerned. That is in regard to sanitation on Federal property. As you all know, there are few communities which do not include one or more Federal buildings. Moreover, there are Customs offices, National Parks, numerous camps established by the Department of National Defence for the use of the army, navy and air force, and, during the war, many munition plants, some of which are still in existence, and munitions storage depots, prisoner-of-war camps and others.

When Federal property is within an organized municipality, the municipal services are used and there is seldom any special action taken to reduce pollution originating on the Federal property. If the municipality has a sewage treatment plant, the wastes are treated; if not, they are discharged with the other wastes. In quite a few instances during the war, it was possible to utilize municipal services if the Federal property was not too far outside the urban area. However, there were hundreds of instances where this was not possible and it was necessary to provide water supplies, sewerage systems and all the other sanitary services connected with urban developments. The Department of Pensions and National Health, at that time the Federal health department, was vitally interested in all such developments, and engineers from the Public Health Engineering Division were constantly in consultation with designing engineers of the other Federal departments, making recommendations regarding the kind of treatment which should be employed for water and sewage, reviewing proposed plans, indeed designing sewage treatment plants in many instances, and finally checking their operation for satisfactory performance. The fact that there were at most only ten or twelve engineers on the staff for all of Canada made conditions somewhat hectic, to say the least. Of course, engineers from the Provincial health departments co-operated on much of this work. It is believed correct to say that, with a few exceptions which had the approval of provincial authorities, all sewage from such projects as have been mentioned received primary treatment and chlorination of the effluent while a good percentage received complete

treatment including chlorination of the effluent. This was not easy to accomplish in view of the shortages of material, and in many instances the primary treatment was the simplest possible; indeed the secondary treatment frequently was developed without mechanical devices, to save metal for other purposes. There were some complaints about nuisances but that was not surprising since many camps, particularly those of the air force, were located near heights of land where dilution of wastes was nearly if not entirely impossible. In some instances the receiving streams were no larger than the flow of sewage effluent.

Sanitation in National Parks is constantly being checked and recommendations for improved installations or more effective operating procedures are made to the Department of Resources and Development, which administers the parks. Similar recommendations are made to the Department of Transport regarding airports under their jurisdiction. The Department of National Health and Welfare has been active in recent years in building hospitals for Eskimos and Indians, and the Public Health Engineering Division has designed sewage treatment plants for them.

A considerable portion of Canada lies outside the boundaries of our ten provinces and is administered by Federal authorities. Here again the Department of National Health is active in restricting pollution of northern waters. In this connection, the sewage treatment plant which was recently put into operation at Yellowknife was designed by the Public Health Engineering Division. This is one of the few such plants in the far north and presented interesting problems in design since it is in the permafrost (permanently frozen) belt of our north country.

Reference might also be made to the National Parks Act, Chapter 33, S. C. 1930, sections 7 (d) and 7 (m), which gives the Federal Government authority as follows:

(d) the management and regulation of fishing; and the protection of fish, including the prevention and remedying of any obstruction or pollution of waterways;

(m) the summary removal from a Park and the preventing of the return thereto of any person guilty of an infraction of such provision of the Park Regulations or the Criminal Code as the Governor in Council may specify;

Also there is the National Harbours Act, under which regulations have been set up to control conditions within the harbour limits. Under the Canada Shipping Act, regulations have been established for the government of public harbours in Canada. These regulations, in section 51, 52, 53 and 54, deal with the pollution of public harbours by refuse, rubbish, oil, etc.

From this rather brief outline, and making due allowance for the limited fields in which Federal action can be taken towards pollution abatement, I feel sure that our efforts in that field will meet your approval. No claim is made that everything possible is being done. In fact, the whole problem is receiving serious consideration, with a view to ascertaining what further action can be taken by Federal authorities. Perhaps the intensive campaign now being organized in the United States will produce worthwhile indications of how we should approach this increasingly important problem in Canada.

Reviewing very briefly the authority under which the Federal Government can control pollution, it is seen that only pollution interfering with navigation is subject to Federal control under the Navigable Waters Protection Act; only pollution destructive to fish can be controlled under the Fisheries Act; only pollution affecting wildfowl, such as oil, may be controlled under the Migratory Birds Convention Act; and there are some regulations under the Canada Shipping Act and the National Harbours Act which make it possible to control pollution in public and national harbours.

Problems of pollution in boundary waters are under the supervision of the International Joint Commission if the pollution crosses the international boundary, to the detriment of health and property on the other side of the boundary.

Sanitation on Federal property is the responsibility of the Federal government and it is here that it can do the most effective work in preventing the degradation in quality of our water resources.

Continued careful supervision will be required in order that all our citizens may be protected and that the best use may be made of our streams and lakes.

Poliomyelitis Virus in Urban Sewage*

AN EXAMINATION FOR ITS PRESENCE OVER A PERIOD OF TWELVE MONTHS

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POLIOMYELITIS virus has frequently been demonstrated in samples of untreated urban sewage obtained during times of epidemic prevalence, and this is accepted as an indication that a large number of persons are excreting virus in the faeces (5, 8, 9, 10, 11).

There is still uncertainty as to how poliomyelitis infection survives in a large urban community from the end of one "season" to the beginning of the next, i.e. in North America from late October or early November through to June. It seems likely, however, that the infection survives in the community by means of the occurrence of a succession of abortive cases or so-called poliomyelitis "minor illnesses". If there is any considerable number of these mild illnesses in the community, virus might be present in the sewage from time to time.

Of particular interest in this connection are studies carried out by Gear (2) in Johannesburg, South Africa, who showed that virus cannot be found until an epidemic has been in progress for several weeks, and that it continues to be recovered for about 2 to 3 months after the close of the epidemic (see also 3, 4, and 7). Tests made in Johannesburg, as well as in the United States, during the interepidemic period, have always proved negative, suggesting that no large number of unrecognized infections occurs in the community (2, 9).

The present study, involving repeated sampling of Toronto sewage over a twelve-month period, was designed to throw further light on this problem of interepidemic survival of infection, as well as to investigate specifically whether Toronto sewage contains poliomyelitis virus at any time.

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Occurrence of cases of poliomyelitis in Toronto During Period of Study

Lists of reported cases of poliomyelitis were obtained from the Medical Officer of Health of the City of Toronto. Each affected residence was checked against a map of the City sewage disposal system to see to which of the two plants it was connected. The Medical Officers of Health of the suburban municipalities were asked to furnish a list of reported cases of poliomyelitis in homes connected to the City system. The few cases thus discovered were added to those occurring in the city.

The date of onset of the first reported case in the area connected to the Main Plant was June 22, 1949. In the area connected to the Northern Plant the date of onset of the first reported case was July 13, 1949.

The monthly incidence of reported cases in the two areas is shown in Table I.

TABLE I

Numbers of Reported Cases of Poliomyelitis in Toronto, and Adjacent Areas Connected to the City Sewage System

Month in which illness commenced (1949)	Residence connected to Northern Plant	Residence connected to Main Plant
June	0	5
July	9	71
August	19	105
September	13	39
October	2	12
November	0	1
Totals	43	233

Toronto Sewage Disposal and Treatment Systems

The City of Toronto is sewered almost entirely on a combined system in which sanitary and storm waters are carried to two treatment works. The Main Sewage Disposal Plant serves approximately 608,800 people and the chief hospitals of the city. We refer to this plant as the Main Plant. The only treatment the sewage receives prior to reaching the point where samples were taken for the present study is passage through racks composed of parallel steel bars spaced 1/2 inch apart, whereby grosser solids are intercepted. Following this treatment the sewage passes to sedimentation tanks prior to discharge into Lake Ontario.

The North Toronto Sewage Treatment Works sewer the northerly section of the city, serving approximately 104,600 people, but only one hospital (for veterans). We refer to this works as the Northern Plant. The only treatment received by the sewage sampled at this plant is passage through coarse racks. Subsequent to sampling, the steps involved in treatment are screening, sedimentation in detritus channels, aeration, and sedimentation in tanks. We have not sampled final effluent from either plant.

Collection of Samples

Sampling was begun in December 1948 and continued until November 1949, samples being collected at approximately 3 to 4 week intervals. At first, collection was only made at the Main Plant but from June 1949 onward additional samples were collected from the Northern Plant.

Samples were collected in glass bottles or jugs holding approximately 4 litres. At the Main Plant, sampling was carried out on each of 3 or 4 successive mornings, at the time of estimated maximal faecal pollution (approximately 9 to 10 a.m.). At the Northern Plant, a similar procedure was followed for the samples collected during June, July, August, and on September 6, 7, and 8. Thereafter samples were collected on each of three or four days at 8 a.m., 12 noon, and 4 p.m. and pooled, thus giving a more representative sampling of the daily flow.

Sewage was held in the laboratory cold room for a few days until prepared for animal inoculation.

Preparation of Sewage for Inoculation

There are considerable technical difficulties in preparing sewage material suitable for monkey inoculation. Many investigators have simply treated the specimen with ether and inoculated the material, comparatively bacteria-free, intra-abdominally. It is preferable, however, to concentrate the contained virus, and ammonium sulfate has been used for this purpose.

Before preparing samples for inoculation, all containers were well shaken and approximately 250 ml of sewage removed from each and pooled. The pooled material was then prepared by one of the following methods:

1. *Etherization.* We have followed a method similar to that used by previous workers (11). The pooled material is shaken vigorously with one-third volume of ether on at least three occasions. When examination discloses that most of the bacteria have been destroyed, the sample is subjected to a vacuum to remove ether, and is then available for intraperitoneal inoculation in monkeys. In certain instances, where bacteria have remained despite frequent ether treatments, we have added penicillin (1000 units per ml) and streptomycin (5 mgm per ml). Previous workers have found that monkeys do not tolerate large quantities of ether-treated sewage, and it has been customary to inoculate amounts of 25 to 100 ml on one occasion. However, we have found it possible to inoculate amounts of 50 ml to 100 ml on successive days and some monkeys in our series have received amounts up to 650 ml in all.

2. *Ammonium sulfate treatment.* Gard (1) introduced the treatment of sewage with ammonium sulfate as a means of concentrating the virus, egg protein being added as a "carrier", and dialysis being used finally to remove excess sulfate. Melnick (5, 6) further improved the method, using monkey serum instead of egg protein and, in the final stage, subjecting the dialyzate to high-speed centrifugation to concentrate virus. We have used the following modification of this technique: About 500 ml of pooled sewage are treated with 10 ml of 2% human plasma in distilled water, and the container is well shaken; then 200 gm of ammonium sulfate are added and shaking is resumed until

the crystals dissolve. After standing overnight in the cold room, the container is shaken, and centrifugation carried out at 3,800 r.p.m. for 1 hour to deposit virus adsorbed to the protein. The deposit is agitated mechanically and resuspended in as small an amount of distilled water as possible (5 to 7 ml). This material is placed in "sausage tubing" with a few sterile glass beads, and dialysis is carried out against cold running tap water for approximately 17 hours (the tap water normally contains free chlorine 0.25 p.p.m.). The dialyzate is then transferred to a double ended syringe, and 1 ml of sterile sodium chloride solution (8.5%) and enough distilled water to bring the final volume to 10 ml are added. One third volume of ether is added and the syringe shaken vigorously. A further ether treatment is sometimes needed. Ether is then removed by exposure to a vacuum. If sterility tests are satisfactory, the material is available for animal inoculation; if not, penicillin and streptomycin are added. Monkeys tolerate well 1.0 ml of ammonium sulfate treated material inoculated cerebrally. Any remaining material, usually from 3 to 4 ml, is injected peritoneally.

Inoculation of Monkeys

Indian rhesus monkeys were chiefly used, but some from Java (cynomolgus) were also inoculated. The complete schedule of monkey inoculations is shown in Table II. Etherized material was inoculated cerebrally (0.4 or 0.5 ml in each thalamus) and peritoneally; ammonium sulfate treated material was injected cerebrally and peritoneally. Our usual practice was to inoculate into the one monkey both material treated by ammonium sulfate and material treated only with ether. Several monkeys succumbed shortly after inoculation due to the toxic nature of some preparations, and have not been considered. Animals were killed as soon as paralysis was noted. Monkeys that did not develop paralysis were mostly killed between 4 and 6 weeks after inoculation. All animals were examined histologically, and the diagnosis is based on the results of this examination.

Results

The results are shown in Table II, from which it will be seen that as regards the Main Plant no poliomyelitis virus was detected by monkey inoculation until the test of samples collected on July 26, 27 and 28. Cases of poliomyelitis in the area connected to this plant were first reported about a month earlier, on June 22. Virus was also recovered from the sewage collected at the Main Plant on August 16, 17 and 18, and September 6, 7 and 8. Virus was not, however, recovered from the samples collected on September 20, 21, and 22, nor from a sample at the end of October. It should be noted (Table I) that the peak incidence of reported cases of poliomyelitis was in August, and that comparatively few were reported in late September or October. In this series of examinations, therefore, we have demonstrated virus to be present during the peak months of July and August and in the early part of September. However, no virus was found in the initial or terminal stages of the epidemic.

The interesting finding in connection with the sampling of the sewage at the Northern Plant was the recovery of virus in the sample collected on June

TABLE II

RESULTS OF EXAMINATION OF TORONTO SEWAGE FOR A PERIOD OF 12 MONTHS

Sample No.	Date of Collection	Plant Sampled	Reference Numbers of Monkeys Inoculated	Type of Inoculum*	Total Volume of Sewage Inoculum (in terms of Unconcentrated Sewage)	Result of Histological Examination of Monkeys
1	Dec. 8, 1948	Main	372	AS	750 ml	Negative
			373	AS	750 ml	Negative
			374	E	140 ml	Negative
			375	E	140 ml	Negative
2	Jan. 3, 4, 5, and 7, 1949	Main	356	AS + E	5250 ml	Negative
			357	AS + E	5250 ml	Negative
			364	AS	3500 ml	Negative
			366	AS	3500 ml	Negative
3	Feb. 28 and Mar. 1, 1949	Main	503	AS	1000 ml	Negative
			504	AS	1000 ml	Negative
4	Mar. 28, 30, 31, and Apr. 1, 1949	Main	528	AS + E	1500 ml	Negative
5	May 2, 3, 4, and 5, 1949	Main	533	E	500 ml	Negative
			544	E	400 ml	Negative
			545	HS	360 ml	Negative
6 A.	June 7, 8, and 9, 1949	Northern	574	E	115 ml	Positive for poliomyelitis
B.		Main	551	E	660 ml	Negative
7	July 6, 7, and 8, 1949	Main	565	AS + E	600 ml	Negative
			576	AS + E	1000 ml	Negative
8 A.	July 26, 27, and 28, 1949	Northern	567	AS + E	900 ml	Negative
			578	AS + E	720 ml	Positive for poliomyelitis‡
B.	July 26, 27, and 28, 1949	Main	577	AS + E	1000 ml	Positive for poliomyelitis
9 A.	Aug. 16, 17, and 18, 1949	Northern	582	E	450 ml	Negative
B.	Aug. 16, 17, and 18, 1949	Main	571	AS + E	800 ml	Positive for poliomyelitis
			603	E	500 ml	Negative
10 A.	Sept. 6, 7, and 8, 1949	Northern	584	AS + E	660 ml	Positive for poliomyelitis
B.	Sept. 6, 7, and 8, 1949	Main	585	AS + E	660 ml	Positive for poliomyelitis
11 A.	Sept. 20, 21, and 22, 1949	Northern	595	AS + E	710 ml	Negative
B.	Sept. 20, 21, and 22, 1949	Main	596	AS + E	710 ml	Negative
12	Oct. 4, 5, and 6, 1949	Northern	619	E	550 ml	Negative

TABLE II—*Continued*

Sample No.	Date of Collection	Plant Sampled	Reference Numbers of Monkeys Inoculated	Type of Inoculum*	Total Volume of Sewage Inoculum (in terms of Unconcentrated Sewage	Result of Histological Examination of Monkeys
13 A.	Oct. 25, 26, and 27, 1949	Northern	625	E	250 ml	Negative
B.	Oct. 25, 26, and 27, 1949	Main	627	E	200 ml	Negative
14	Nov. 15, 16, and 17, 1949	Northern	642	E	360 ml	Negative
15	Nov. 28, 29, 30, Dec. 1, 1949	Northern	641	E	360 ml	Negative

*AS = Ammonium sulfate treated.
E = Ether treated.
‡Suspension of C.N.S. of monkey 578 induced typical poliomyelitis in monkey 583.

7, 8, and 9. No reported cases occurred in the predominantly residential area served by this plant until 5 weeks later (July 13). The recovery of virus was made about 2 weeks before the first case reported in the area of the City connected to the Main Plant. Virus was recovered from the Northern Plant also at the end of July, and again in early September. All subsequent samples have, however, proved negative.

Survival of Virus in Stored Sewage. It is of interest to record that three of the samples giving positive results in monkeys were stored for some weeks in the refrigerator before monkey inoculation. This was necessitated by the fact that monkeys inoculated with material prepared shortly after collection died from non-specific causes. Thus sample 6A, collected on June 7, 8, and 9, was inoculated in a monkey 90 days later and induced typical poliomyelitis. In the case of samples 8A and 8B there was a period of 47 days' storage before the monkeys were inoculated.

DISCUSSION

Our finding of virus in the sewage of the Northern Plant substantially before the recognition by physicians that poliomyelitis was prevalent in the area, affords some support for the suggestion that poliomyelitis virus becomes widely disseminated in a community in the period immediately before the occurrence of frankly paralyzed cases. No isolation of virus has yet been made in the winter months, and it is probable that the number of persons excreting virus at this season is very small. Examinations are, however, being continued, and the sewage from the Northern Plant is being sampled at intervals of approximately two weeks.

With regard to technique, we have found that the peritoneal inoculation of etherized sewage in monkeys in amounts of 50 ml daily for 5 or more days is

very satisfactory. Somewhat larger amounts can be tested if the ammonium sulfate method of concentration is adopted. This procedure, however, probably involves some destruction of virus, and should not be relied upon as the only method of examination. It is hoped shortly to investigate the preparation of samples by ultracentrifugation in the Spinco ultracentrifuge, where amounts of 360 ml can be spun at one operation (13).

Finally, it is of interest that virus can survive in sewage stored in the refrigerator for at least 90 days. These observations are similar to those we have already made on the survival of virus in artificially infected river water (12).

Summary

1. An examinaton of sewage collected at the two plants of the City of Toronto has been conducted regularly for about 12 months.
2. Virus was recovered from both plants at the height of the epidemic in July, August, and September 1949.
3. Virus was isolated from sewage collected at one of the plants which serves a residential area in June 1949, 5 weeks before the first reported case of poliomyelitis in the district.
4. It has been found that monkeys will tolerate up to 500 ml of etherized sewage, if inoculated intraperitoneally in daily amounts of 50 ml.
5. Virus in sewage has been shown to survive in the refrigerator for at least 90 days after collection.

Acknowledgements

I am indebted to the Commissioner of Works, City of Toronto, for kindly supplying details of the sewage disposal and treatment plants, and to Messrs. Nevitt and Bonham under whose direction the samples were obtained. The Medical Officer of Health for the City of Toronto kindly furnished us with a list of reported cases of poliomyelitis.

Drs. A. F. Graham and R. C. French kindly carried out many of the "runs" in the ultracentrifuge.

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Plague

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AS long as headlines such as "Black Death Stirs Albuquerque," "Black Plague Hits Two in New Mexico," etc., occur, it is necessary that we who are interested in public health and preventive medicine should acquaint ourselves with plague as it occurs today, including its prevalence and its potentialities.

To understand and follow the spread of a communicable disease, it is necessary to be able to demonstrate the seed, the sower, and the soil. We have such information relative to bubonic plague, which is characterized by inflammation and enlargement of lymph glands—axillary, cervical, and inguinal—and pneumonic plague, which is characterized by respiratory symptoms, capillary hemorrhages, and frequently by sudden death. In the minds of investigators, bubonic plague had long been associated with domestic rats, and in 1907 the Indian Plague Commission found the rat flea to be the vector transmitting the infection from the rat to man. It was not until 1894 that Kitasato and Yersin, working independently, isolated and described *Pasteurella pestis*, the organism producing plague. If the domestic rat were the only reservoir of infection, then improved standards of living and environmental conditions aimed at breaking the connection between man and rat would render the spread of plague improbable or even impossible.

Careful study of the history of plague would seem to indicate that the disease centered around the Central Asiatic Plateau. Pandemics stemming from this area would subside for as long as two centuries and then occur in another area, again stemming from central Asia. Each pandemic was associated with an epizootic among domestic rats. The first reference made by modern medical literature to plague in wild rodents was in 1895 when Bjeliavski described this infection in the tarabagan, the marmot of Asia. However, it is known that the Mongols possessed knowledge of plague, which they passed from generation to generation together with legends dealing with the tarabagan and its dangers. These considerations give strength to the theory that central Asia is the foci of plague and that the disease is perpetuated among the wild rodents, more particularly the tarabagan.

The first case of plague diagnosed in the United States was that of a Chinese in San Francisco, California, March 6, 1900. Previous to this, plague

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had been found on ships at quarantine stations in the United States: Port Townsend, San Francisco (1899), and New York. There are two theories for the source of this first case of plague in San Francisco: (a) infected rats from ships arriving from the Hong Kong area where a pandemic had been in existence since 1894, or (b) infected wild rodents that had entered this continent by way of the Aleutians with the original settlers. Between 1900 and 1904, San Francisco had 120 cases of plague with 114 deaths. These were in Chinatown, and an epizootic among domestic rats was in progress at the same time. A drastic rat eradication program was conducted, and no more cases were reported until 1907. The great earthquake and fire of 1906 had readied rat harborage and breeding places. During 1907, 178 cases with 87 deaths were reported. These cases were scattered over San Francisco and surrounding areas. The State of Washington had three cases with three deaths during the same year.

In 1908 plague was demonstrated in ground squirrels in Contra Costa County, which is near San Francisco. This was the first proof that the infection had spread to wild rodents in California and was the beginning of the study of plague infection of wild rodents by the U.S. Public Health Service. The study started in California and has continued through all of the western states. The survey teams of the U.S. Public Health Service have covered territory reaching to the 100th meridian, which passes through the United States only a short distance west of the geographic center and is almost parallel with the eastern border of the panhandle of Texas. In the beginning, wild rodent infection was demonstrated by inoculating portions of the organs of possibly infected rodents into guinea pigs. In 1936 C. R. Eskey adopted the method of parasite inoculation of guinea pigs. This method permitted more rapid examination of rodents with less possibility of secondary infection of the test animals.

Since 1900 there have been 515 cases of plague in the United States, with 338 deaths. In the last 25 years, there have been 25 cases with 13 deaths—not a very drastic picture when one compares that record with that of many other communicable diseases. Since there was on the average only one case per year during the last 25 years, it would seem that the problem is well under control until one studies the reports of the U.S. Public Health Service and learns of the rapidly expanding areas in which plague-infected rodents and ectoparasites have been found. Within 10 years after plague first appeared in San Francisco, plague had been demonstrated in wild rodents in nine surrounding counties. By 1935, plague-infected wild rodents were found in Montana and Oregon, and in 1938 in New Mexico. Since 1908 infected rodents have been found in 15 western states. The evidence and records would indicate that plague spread from domestic rats in San Francisco first to the ground squirrels and then to other wild rodents in the western states. This spread of plague in wild rodents has gradually extended from the Pacific coast eastward and has now been demonstrated in those states cut by the 100th meridian. Any attempt to eradicate wild rodents would fail; hence we must accept the fact that we have in the wild rodents in the United States a reservoir of plague infection. As long as the reservoir of infection is in the wild rodent, we could

expect only a few cases and those widely scattered unless a pneumonic infection should occur, when rapid spread might be expected.

The New Mexico Public Health Laboratory has been on the alert for cases of plague since 1938 when the U.S. Public Health Service first reported plague in the wild rodents of one of our counties. There was no suggestion of human infection until August 1, 1949, when two cases occurred.* The two cases were from areas 200 miles apart with no connection. One case, a boy of Taos, probably received his infection from the bite of a flea escaped from the body of a prairie dog which he killed by throwing a stone. The fact that he could be close enough to kill the animal with a stone indicated that it was sick. The second case, a man from Placitas, probably received his infection from fleas from ground squirrels that he had killed while working on his farm. The man showed skin lesions on the thigh that were probably due to the bite of the flea. The child was critically ill when first seen by his physician. He received sulfadiazine and streptomycin and made a rapid, uneventful recovery. The man was moderately ill when admitted as a patient at the Veterans Hospital. His medication included sulfadiazine and penicillin, and his recovery was uneventful. (Statements relative to medication were given by telephone by the physicians in charge of the cases.)

These two New Mexico cases of human plague are good examples of what might be expected considering the reservoir of plague infection in the wild rodents of the western states.

As plague in wild rodents spreads eastward—and it is spreading rather slowly but steadily—there will be more and more possibility of domestic rats becoming infected from wild rodents. If an epizootic of plague in domestic rats was established, many human cases might occur although modern methods of sanitation and of medical care would prevent a repetition of the pandemics of history.

What can be done to prevent the spread of plague? Infected wild rodents cannot be eradicated. Active measures should be taken to decrease the domestic rat population. This should include rat proofing of buildings and the elimination of sources of a food supply for the rats. Scientific garbage and refuse disposal is indicated as well as better storage of grain and other agricultural products. Since the disease is transmitted to man from the infected rodent by the ectoparasites of the rodent, a break in the chain may be made by killing the parasites on the rat. This may be accomplished in part by dusting rat runways and rat harbors with an insecticide such as DDT.

We probably will continue to have isolated cases of plague among those who frequent areas populated by wild rodents. Adults and children should be educated relative to care necessary to avoid contact with the wild rodent and his parasites and particularly to be on the alert for sick animals, for it is

*Since this paper was submitted, two additional cases of bubonic plague have occurred in New Mexico. On November 5, 1949, a boy from San Patricio, Lincoln County, died of plague, the case not being diagnosed until after death.

On January 14, 1950, laboratory specimens were received from a man from Maljamar, Lea County, a patient at the Veterans Hospital, Albuquerque. *Pasteurella pestis* was isolated from the specimens, and the patient made an uneventful recovery. This man had skinned wild rabbits. Later the U.S. Public Health Service determined that cottontails in this area were sources of plague.

the parasite from a sick animal or the sick animal's nest that is prone to be an infective parasite. Not all infected parasites are infective.

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AN APPRECIATION OF DR. JOHN T. PHAIR

THE thirty-eighth annual meeting of the Canadian Public Health Association, held in Toronto during the second week of June, afforded the members an opportunity to pay tribute to the contribution made to the organization over the years by the president, Dr. John T. Phair, Deputy Minister of Health and Hospitals for the Province of Ontario.

For nearly four decades, the Association has been unique in that it has conducted its program without senior salaried executive officers, with the exception of a period of three years when it had the services of Dr. J. H. Baillie to conduct special field studies. For twenty-four of these years, Dr. Phair served the Association as Honorary General Secretary, having assumed this post at the annual meeting in Saint John in 1922. Although his responsibilities in the Department of Health of Ontario increased from year to year, he continued to find time, because of the need, to guide the Association's policies and program until 1945, when his appointment as Deputy Minister of Health for the Province made it impossible for him to serve any longer as General Secretary.

The Association's annual dinner, held on Tuesday, June 13th, gave the members an opportunity to express their appreciation of his services through the years. On behalf of the Association a former president, Dr. F. W. Jackson, of the Department of National Health and Welfare, presented Dr. Phair with an address which had been beautifully illuminated by Miss Vera Crossley, a senior member of the Central Laboratories of the Department of Health of Ontario. The text of the address, reproduced below, expresses something of the gratitude which the members of the Canadian Public Health Association feel.

To

JOHN THOMAS PHAIR

M.B., D.P.H.

THE Members of the Canadian Public Health Association, at the thirty-eighth Annual Meeting held in the City of Toronto on June thirteenth, nineteen hundred and fifty, express to you their appreciation of the services which you have so generously contributed during the past twenty-eight years.

In nineteen hundred and twenty-two, when you assumed the duties of Honorary General Secretary, the Association's future was uncertain. To maintain a national

public health association, active support was required from all public health workers in Canada, as well as from the Federal and Provincial Governments. Today the Association enjoys that support, and to you it owes a special debt of gratitude for your leadership.

When you assumed the post of Chief Medical Officer of Health for the Province of Ontario in nineteen hundred and thirty-five you continued your active interest in voluntary health agencies, and in addition to serving as General Secretary of the Association you became honorary adviser in public health to the Canadian Red Cross Society and chairman of the National Junior Red Cross Committee; you continued as medical consultant to the Victorian Order of Nurses for Canada; and you gave generously of your time to the Conference of State and Provincial Health Authorities of North America. In nineteen hundred and forty-five, when you became Deputy Minister of Health and Hospitals for the Province, it was regretfully realized that your increasing responsibilities would make it impossible for you to serve as Honorary Secretary of the Association.

Your twenty-eight years of service in a voluntary agency has no equal in Canada. Its reward has been solely the satisfaction of seeing the development of a strong national public health association. Because of the Association's need, you gave your best; because of your ability, the Association has succeeded.

To you, the members of the Association express their heartfelt thanks and extend their best wishes for your continued success in the field to which you have devoted your life.

THE RISING TIDE OF POLIOMYELITIS

IN CONTRAST to the general reduction in the incidence of the communicable diseases of childhood, venereal disease, and tuberculosis, there has been a steadily increasing number of cases of anterior poliomyelitis in the United States during the past decade. In the report of the National Foundation for Infantile Paralysis for 1949, it is pointed out that 42,375 cases were recorded—the highest number ever reported. In the first six months, barely 3,000 cases were reported. By the middle of July, more than 1,000 new cases were being recorded in a single week. The weekly figure increased to 2,000, and by the middle of August a peak of 3,415 cases had been reached. A striking poster entitled "The Rising Tide of Poliomyelitis" presents the mounting incidence from 1938 through 1949.

These facts should be in the minds of public health administrators throughout Canada. Canada has not suffered to the same extent in this period as the United States, but we have had many serious outbreaks. Intensive work is being done in many centres to learn more of how the disease is spread and particularly to evolve effective methods of control. Research, however, can be conducted only in centres which are properly equipped for adequate investigation of this disease and staffed by competent investigators. The control of poliomyelitis awaits the development of methods based upon these researches. The problem of poliomyelitis is of increasing magnitude.

NEWS

British Columbia

SCHOOL DISTRICT NUMBER 42 and the Municipalities of Maple Ridge and Pitt Meadows are joining the North Fraser Valley Health Unit, which was opened in October 1949. The North Fraser Valley Health Unit is under the direction of Dr. R. T. M. Puddicombe, who joined the Provincial Department of Health in the fall of 1949. Dr. Puddicombe received his M.R.C.S.(Eng.) and L.R.C.P.(London) at St. Thomas' Hospital, London, England. Before joining the Department, he was with the Leduc Clinic in Alberta.

DR. J. A. TAYLOR and Dr. G. R. F. Elliot attended the meeting of the Western Branch of the American Public Health Association held at Portland, Oregon. Dr. Elliot presented a paper on cancer control in British Columbia and while there Dr. Taylor met with officials of the University of California, State health officers, officials of public health schools and others to discuss recruitment and post-graduate training.

DR. CHARLES L. HUNT, Director, Division of Venereal Disease Control, and Mr. John Doughty attended the annual meeting of the Canadian Public Health Association in Toronto. Dr. Hunt and Mr. Doughty presented papers in their respective sections.

Dr. H. M. Brown was appointed to the Cariboo Health Unit to succeed Dr. T. H. Paterson, who will be leaving for post-graduate training this fall.

SICKNESS SURVEY plans are being completed for the survey of morbidity throughout British Columbia. It had been hoped to get this started by June 1st but preparations could not be completed in time so that postponement to a date in early fall is now considered to avoid the interruptions anticipated from summer holiday schedules.

Saskatchewan

IN ADDITION TO the Branches of Preventive Services and Administrative Services, four others were established during the recent reorganization of the Department of Public Health. The Medical and Hospital Services Branch includes the Saskatchewan Hospital Services Plan, the Division of Hospital Planning and Standards, the Division of Hospital Accounting and Administration, the Medical Services Division and the Air Ambulance Service, and will supervise Municipal Doctor plans. The Research and Statistics Division becomes a branch and includes the Public Health Library. There will be a direct functional relationship between this Branch and the Health Services Planning Commission, which now reverts to its original function as a planning body. The Regional Health Services Division has been raised to branch status, and the Mental Services Division becomes the Psychiatric Services Branch.

BECAUSE OF the need for trained physiotherapists in Saskatchewan, the Department of Public Health has established three bursaries of $1,500 each to provide for one-year post-graduate courses in physiotherapy. The one-year course leads to a certificate and membership in the Canadian Physiotherapy Association, and will qualify persons for positions now open in Saskatchewan hospitals. Training may be taken at American Schools of Physiotherapy approved by the Canadian Physiotherapy Association.

A FOUR-MONTH TICK and plague survey of Saskatchewan, approved by Ottawa under the Federal Health Grants, is now under way. This is a co-operative venture between the Department of Public Health, the University of Saskatchewan, and the Dominion Government Laboratory at Kamloops, B.C. The purpose of the survey is to search for ticks infected with the virus of Rocky Mountain spotted fever and small animals infected with plague. It has been planned carefully with a view to providing a basis for a comprehensive survey in ensuing years. The field work is being done by a two-man crew using a mobile laboratory. This survey is being undertaken merely as a precautionary measure as, to date, no human cases of spotted fever or plague have been diagnosed in the province. The findings will be correlated with those of Alberta, British Columbia, and the United States Public Health Service.

MR. GERALD C. STEWART, of the Division of Hospital Planning and Standards, has been awarded a one-year fellowship in hospital administration by the University of Rochester Medical School and the Strong Memorial Hospital at Rochester, N.Y. Mr. Stewart, who holds a B.Comm. degree from the University of Saskatchewan, has been on leave from the Department for the past year to take the course in hospital administration at the University of Toronto. His work in Rochester will complete the requirements for a Diploma in Hospital Administration.

Manitoba

DURING the recent disastrous flood in Manitoba, relief and special assistance were offered from all parts of Canada. Among the special personnel employed in the flood control program were doctors, veterinarians, engineers, and sanitary inspectors.

One veterinarian from outside the province was Dr. H. Bryson of Vancouver. The engineers included Messrs. R. Bowering, Victoria; A. Perry, Vancouver; R. Comstock, Regina; and G. M. Galinbert, D. Stokoe, and O. Ball, all of Toronto.

Almost 50 sanitary inspectors participated actively in flood control work. These included: N. L. Bruce, Calgary; H. Dean, Lethbridge; L. Stewart, Edmonton; J. McGoey, Regina; A. Ball, Prince Albert; S. Price, Moose Jaw; L. Sitler, Saskatoon; F. Symons, Regina; H. Tyler, A. O'Hara, A. Ruggles, W. McKenzie, R. Welsh, T. Neil, B. Richardson, E. Cook, C. R. Kennedy, W. G. Cooke, all of Toronto; D. Wood, Kirkland Lake; R. Hall, Northumberland-Durham Unit; H. Terry, St. Catharines-Lincoln Unit; I. Rose-Christianson, Department of Agriculture; F/Sgt. J. Crate, R.C.A.F., G. E. Mitchell, B. Stephen, J. S. Puls, Dauphin; A. Symko, Kildonan-St. Paul; T. S. Johnson, Neepawa, F. K. Collins, Flin Flon; C. H. McEwen, Portage la Prairie; J. Sicotte, Steinbach; W. Fedak, J. J. Courteau, St. Boniface; L. J. Hunter, St. James; G. L. Swain, St. Vital; L. King, G. J. Philips, Selkirk; A. E. Lunde, Stonewall; A. W. Kay, Swan River; J. H. Broomhall, Virden; and H. R. Christianson and W. F. Noyes, Winnipeg.

In addition to the people mentioned on the list, the permanent staff at Central Office were engaged almost full-time on this emergency. Dr. Hugh Malcolmson co-ordinated the general administration; Mr. W. M. Ward, chemist, took over control and distribution of chemicals, deodorants and disinfectants; Mr. M. Flattery, senior inspector, was general supervisor of activities in the Greater Winnipeg area; Mr. K. G. MacQuarrie supervised all activities in the so-called "Valley of the Red" between Emerson and Greater Winnipeg; Dr. E. C. Chamberlayne, Director of Food Control, was in charge of food; and the work of the visiting sanitary engineers was co-ordinated by Mr. L. A. Kay.

DR. CARL BUCK, of the School of Public Health, University of Michigan, was in Manitoba in May on his second survey of the Province's health facilities. Dr. Buck was asked to assist members of the Advisory Health Survey Committee to bring up to date his 1941 report which led to the passing of the Manitoba Health Services Act of 1945. Dr. F. W. Jackson, Director of Health Insurance Studies for the Department of National Health and Welfare, was in Manitoba during Dr. Buck's survey. Dr. Jackson was Deputy Minister of the Manitoba Department of Health and Public Welfare at the time of Dr. Buck's first survey and the passing of the Health Services Act.

DR. F. R. CHOWN, Medical Director of the Selkirk Health Unit, is assisting the members of the Manitoba Advisory Health Survey Committee who are preparing their report for the Federal Government in connection with the program of health grants. Dr. J. M. O'Keefe is acting Medical Director of the Selkirk Health Unit.

DR. V. S. HAWKES, Medical Director of the Stonewall Health Unit, and Dr. E. Mastromatteo, Medical Director of the Virden Health Unit, returned to their posts after successfully completing their studies for the Diploma in Public Health at the University of Toronto.

DR. C. MANLY has been transferred from his post as Medical Director of the Portage la Prairie Health Unit to Medical Director of the Northern Health Unit with headquarters at Flin Flon.

THE NEW MINNEDOSA DISTRICT HOSPITAL, with 27 beds and 12 cubicles, was officially opened April 24th. The Swan Valley District Hospital, which has 30 beds, was opened May 24th. This new building will house the Swan Valley Health Unit.

A NURSE RECRUITMENT CAMPAIGN WAS

held in Manitoba in May and June to encourage high school students to take up the career of nursing. Mrs. Julie Beatty, R.N., and Miss Margaret Kennedy addressed groups in 33 towns and cities in Manitoba. The nurse recruitment committee was set up by the Manitoba Advisory Council for Health Services and its members represent four associations: the Manitoba Association of Registered Nurses, the Manitoba Association of Licensed Practical Nurses, the Manitoba Association of Hospital Aids, and the Manitoba Hospital Association. An essay contest was held as part of the campaign and prizes were presented to the winners at the Manitoba premiere of the documentary film "Challenge—Science Against Cancer". A 20-year-old Cree Girl, Miss Jessie M. Ross from the Indian Residential School in Brandon, received first prize of $75.00.

DR. J. M. BROWN resigned as Medical Director of the Red River Health Unit to take a post in Tranquille Sanitorium, Kamloops, British Columbia.

THREE MEMBERS of the Manitoba Department of Health and Public Welfare attended the June Conference of the Canadian Public Health Association in Toronto. They were: Miss Margaret E. Nix, Director, Health and Welfare Education, Dr. M. R. Elliott, Director, Extension Health Services, and Mr. L. A. Kay, Public Health Engineer.

THE DOCUMENTARY FILM, "Challenge—Science Against Cancer", was shown in Winnipeg, Brandon and Dauphin in April and May to more than 2,000 people.

RATEPAYERS in the 13,000 square miles around Virden, Manitoba, will vote June 27th on the establishment of a new hospital district. The plan calls for a new-30-bed hospital to be built in Virden, replacing the present hospital which was built in 1910. Medical-nursing units will be built in the towns of Reston and Elkhorn and a nursing unit is also planned for the town of Oak Lake.

Ontario

MISS MARION V. PENRICE, a graduate of the University of Toronto, 1948 (Household Economics), and of the Ontario College of Education, 1949, has joined the staff of the Provincial Department of Health. Miss Penrice holds a High School Assistant's Certificate in Home Economics and obtained the Certificate in Public Health from the School of Hygiene, University of Toronto, during the 1949-50 session.

New Brunswick

THREE MEMBERS of the New Brunswick Department of Health medical staff have returned to the province recently after completing work on advanced training in special fields. Dr. A. F. Chaisson is back in Fredericton where he had made his central headquarters as District Medical Officer for York, Carleton and Victoria Counties before going to the University of Toronto, School of Hygiene, a year ago. He has received his D.P.H. and is attached to the office of Dr. A. M. Clarke, D.P.H., Assistant Chief Medical Officer and Director of Health Planning Services.

With the greater portion of the provincial health survey now completed, Dr. Chaisson is summarizing and consolidating the great fund of material gathered under the survey. He will prepare this information for submission to Ottawa late this summer in connection with briefs to be placed before the fall Dominion-Provincial Conference by New Brunswick. About 18 subjects are being covered.

Dr. C. R. Trask, Saint John, and Dr. J. R. Allanach, Woodstock, District Medical Officers, are back at their provincial posts after completing courses in diagnosis and treatment of tuberculosis at the Trudeau Foundation, Saranac Lake, New York State.

Remarkable results have attended the semi-annual conferences held by New Brunswick Department of Health staffs in the past year or two. The late spring sessions of the Public Health Nursing Service, called by Miss Muriel Hunter, R.J.G.N., its director, drew together the largest group since this program was started. There are 19 nurses in this Division now as the program continues to expand. These meetings lasted four days.

Some of the new drugs now being used were discussed by Dr. H. H. MacKinnon of Fredericton when he spoke before the public health nurses. Another local physician and surgeon, a specialist, Dr. W. Ross Wright, Fredericton, spoke on eye defects found in infants and pre-school and school children. The relation of mental health to the public health nurse in the field was explained by Dr. R. R. Prosser, Director of Mental Health with the Department. Other speakers from the Department included Dr. Ruth McDougall, Director of the Maternal and Child

Health Division; Miss Florence Swan, Senior Nutritionist, and Miss Katherine MacLaggan, Assistant to the Director of Public Health Nursing and Director of the Health Program at Teacher's College.

Dr. J. A. Melanson, Chief Medical Officer, presided over the two-day sessions of the District Medical Officers. A wide variety of subjects was taken up, with the most emphasis on hospital facilities, laboratory services, immunization schedules, infant mortality, crippled children, nursing services, sanitary engineering and services, restaurant conditions, the handling of dairy products, progress in the tuberculosis field, uniformity of reports and the health survey program.

New Brunswick's first out-patient neuropsychiatric clinic has been opened at Saint John General Hospital. It is held twice weekly at present under the direction of Dr. R. A. Gregory of the Provincial Hospital staff. One psychiatrist, one clinical psychologist, one psychiatric social worker and the clerical staff form the psychiatric team at the clinic. It accepts all cases referred to it by doctors and certain social agencies. The purpose of the clinic at first instance is to offer a diagnostic and consultation service to all practising physicians. Certain types of patients will receive treatment.

The Hon. Dr. James W. Brittain, LL.D., Minister of Education and Municipal Affairs, is acting as Minister of Health and Social Services while the Hon. F. A. McGrand is out of the Province.

BOOKS

Photo-Radiography in Search of Tuberculosis.

By David W. Zacks, M.D. Baltimore: The Williams & Wilkins Company, 1949. Canadian agents: The University of Toronto Press, Toronto 5. 297 pp. $5.00.

PHOTO-RADIOGRAPHY, mass radiography or miniature radiography has become, with justification, one of the most potent and important factors in the efforts to control tuberculosis. Certainly, no plan can be considered as completely effective without the assistance of this valuable method of examination.

Those who have had experience with this method of case-finding must have been impressed with the numerous problems which are created by the procedure. This makes the book by David W. Zacks, "Photo-radiography in Search of Tuberculosis", a much appreciated contribution and a serious and sincere endeavour to ferret out the answer to many of the perplexing questions which arise in the course of mass radiography. The fact that Dr. Zacks writes with a background of 20 years of "service" adds to the value of his observations.

The author cautions that enthusiasm for mass radiography must be tempered by the fact that positive findings indicate, not the answer, but the beginning of a series of questions seeking answer. In the principles which he applies to the search for such answers he shows his intimate experience with the value of the procedure, provided corollary investigational facilities are available.

The book has 278 pages and can be divided into 3 sections. The first section of 98 pages presents numerous observations dealing with principles and details of approach to and organization for mass radiography. The information is carefully organized and concisely presented, containing answers to virtually any problems which may arise. The second section consists of illustrations of 14 typical cases of tuberculosis and an atlas of chest photo-radiograms. This section of 129 pages, carefully chosen, is a most satisfactory range of problems in x-ray shadows. Unfortunately, reproduction of x-rays on paper still leaves much to be desired. The third section contains specimens and good suggestions about promotional and educational material. A satisfactory bibliography completes the volume.

The book is attractively bound and of convenient size. It is a volume which well deserves a place in the much-used books section of the library of any organization interested in the search for tuberculosis.

C. J. W. Beckwith

Industrial Toxicology.

By Lawrence T. Fairhall. Baltimore: The Williams & Wilkins Company, 1949. Canadian agents: The University of Toronto Press, Toronto 5. 483 pp. $6.00.

FAIRHALL'S NEW BOOK is a welcome addition to the literature of a field which until rather recently was poorly supplied with adequate text-books. Since the volume has been designed as a reference manual for the industrial hygienist, emphasis is placed on practical aspects rather than on the more academic questions such as fundamental mechanisms by which toxic effects are produced. Some seventy-five inorganic and one hundred and thirty-five organic compounds have been selected for examination. A description of the characteristic properties and industrial uses of each compound is provided, along with information on toxicity and analytical procedures suitable for field and clinical use. The compilation of physical constants and important reactions of each compound is a very useful feature, and of particular value are the data subsumed under industrial uses. The presentation of information on each substance is uniform and consistent; the discussions of beryllium and cadmium as industrial hazards and on the toxicology of trinitrotoluene are especially noteworthy. Interesting sections are devoted to the chemistry and toxicology of plutonium and of radio-active substances.

In addition to the body of the text, there is a short introduction which briefly treats the topics of the forms in which hazards occur, the relations between chemical constitution and physiological response, and the

experimental approach to toxicology. It is unfortunate that mercury was chosen to exemplify the physical systems, dusts, fumes and gases, since these were all incorrectly defined. The reviewer cannot agree with the author that such systems are not susceptible of exact definition.

While the applied and practical treatment of the subject matter limits the value of this book as a teaching text, it should prove of value as a reference work not only to the practising industrial hygienist but also to the industrial physician.

H. N. MacFarland

Textbook of Virology.

By A. J. Rhodes, M.D., F.R.C.P.(Edin.), and C. E. van Rooyen, M.D., D.Sc., M.R.C.P.(Lond.). New York and Toronto: Thomas Nelson & Sons, 1949. 312 pp. $5.50.

WHILE EXCELLENT TEXTS on particular aspects of virus infections and detailed accounts of the common communicable virus diseases are readily available, the student or practitioner wishing to survey the general field of virus and rickettsial infections has, too often, been forced to search out the material from a multiplicity of sources or alternatively to consult larger treatises covering the subject in detail well beyond his needs. Usually he finds time to do neither. This book, which presents a concise review of the present knowledge of the wide range of virus and rickettsial diseases now recognized, is therefore a timely contribution.

The presentation, which has been evolved from courses of lectures delivered to students over a period of years, is designed as an introduction to the subject in a form and extent deemed suitable for medical students and practitioners. While undue detail is avoided, the essential features of value to the student are adequately discussed.

The early chapters deal with the general properties of viruses. A working classification of virus diseases, grouped according to the tissue affected and clinicopathologic features, is adopted and followed in the latter part of the text. The nature and fundamental properties of viruses, virus immunity mechanisms, and the mode of spread of virus diseases are discussed. Technical methods and apparatus employed in virus studies are outlined and the common methods described in detail.

Succeeding chapters are devoted to an orderly discussion of individual virus infections. In each case a short description of the salient clinical features and a brief account of the pathology is followed by more extensive treatment of the properties of the virus, the epidemiology of the disease, diagnostic laboratory procedures where applicable, and methods of prevention.

The final chapters are concerned with the biology of the Rickettsiae and a description of the rickettsial diseases of man in a pattern similar to that employed for the virus diseases.

R. J. Gibbons

An Introduction to Parasitology.

By Asa C. Chandler. New York: John Wiley & Sons, Inc., 1949, 8th ed. 756 pp. $6.00.

THE EIGHTH EDITION of this book is largely rewritten, although the previous arrangement has been maintained. The text is divided into three parts, Protozoa, Helminthology, and Arthropods, and sub-divided into twenty-eight chapters. A short list of useful references follows each chapter and a number of leading journals are cited at the end of the book.

The book is brought up-to-date with discoveries and advances in the field of parasitology since the last edition was published five years ago. Much new material has been added to the chapters dealing with arthropod-borne diseases, insecticides, repellents and their uses. The parasites of man are fully discussed and common parasites of domestic and other animals are included. Several new keys for identification have been added. Life cycles of parasites, epidemiology, general principles of treatment and prevention of parasitic diseases, and host-parasite interrelations are well presented.

The book gives a comprehensive account of parasitology and is recommended as a textbook in the courses of general parasitology. Medical students, nurses, and laboratory technicians will find it equally useful.

Ella Kuitunen-Ekbaum